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ALLERGIC DESENSITIZATION BY THE INTRADERMAL ROUTE

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The subcutaneous route is and has been used almost universally for the past 50 years for the administration of pollen and other extracts in allergic desensitization procedures. The intradermal* method, however, is the one almost exclusively used in South Africa.

Following upon my recent description[1] of patients who are sensitive to bee stings, in whom effective desensitization had been achieved by the use of gradually increasing strengths of whole bee extract administered by the intradermal route, I have received numerous enquiries for details of this method. It is aimed here to describe the principles, procedures and techniques involved and, not the least, to make better known the advantages of this eminently satisfactory and safe method of desensitization.

There is considerable experimental and clinical justification for immunization *via* the skin. According to Kahn[2] the antigen-localizing capability of the cutaneous tissue is approximately 10 times as great as that of skeletal muscle and is evidently a highly important defensive mechanism against micro-organisms; furthermore, the skin has a reaction-capacity for allergens above that of most other tissues.[3] Rappaport[5] referred to the many authenticated experiments which emphasized the importance of the skin as an active immunologic organ and was of the opinion that the skin of atopic patients contains specific reaginic antibodies in all epidermal cells. Tuft[6] thought that the skin was an extremely important immunologic organ participating very actively in antibody production and that, in certain infectious diseases (small-pox, chicken-pox, measles, and scarlet fever), cutaneous antibody formation was the means by which immunization of the whole body was brought about. He explained the marked antibody response which he uniformly obtained with the intradermal administration of mixed typhoid vaccines as resulting from a local tissue stimulation of the cells of the reticulo-endothelial system, which were particularly abundant in the skin, and, in part also, to a slower absorption of antigen which allowed for local fixation of a larger amount of antigen —and thus for a greater local stimulation of antibodies. In a review of the evidence for the immunologic importance of the skin, Sulzberger[7] concluded that the skin was probably the organ principally concerned in the mechanism of immunologic protection and of allergic alteration. Walzer[8] also regarded the skin as one of the most valuable organs of the body in its immunizing properties. Müller and Corbitt[10] showed that the effect of insulin injections upon the blood-sugar content of the normal rabbit varied with the route of administration and was markedly increased by the employment of the intradermal route. They thought that the degree and period of effectiveness did not depend upon the rapidity of resorption, but upon a hitherto unknown factor which was probably related to the action of the involuntary nervous system—a property seemingly inherent in the skin rather than in the subcutaneous tissue or in the body fluids. Elaborating on this 'special function' of the skin, they pointed out that 0·1 ml. of aolan, a non-specific lactalbumin preparation, was an effective stimulant of the involuntary nervous system if given intradermally, but that no measurable change occurred in the organs controlled by this system after the administration of the same quantity of this agent by other routes. Smith[11] described the intradermal injection as essentially an intralymphatic injection which produced effective antigenic stimulation of the lymph node, an easily accessible and clinically useful site.

The use of the intradermal route for purposes of immunization has been frequently reported in connection with the administration of a variety of antigenic substances—diphtheria,[12,13] scarlet fever,[14,15] mumps,[16] typhoid fever,[17-19] influenza,[20-26] tetanus,[27] poliomyelitis,[28] and rabies.[29]

The beginnings of desensitization *via* the skin are seen in the work of Le Noir *et al.*[30] who reported 5 instances of sensitization to certain foods in which the application of the specific substance to the skin induced a general anaphylactic reaction either immediately or after an interval of 1—24 hours. This suggested to them the possibility of accomplishing desensitization by repeatedly eliciting the skin reaction. Similarly, Vallery-Radot and Blamoutier[31] held that the repeated application of minute quantities of the incriminated protein, merely for a *diagnostic* reaction, was a simple and harmless procedure for desensitizing against asthma, urticaria, and eczema. Thommen[9] noted a 'somewhat common experience'—that the acute symptoms of patients were often definitely alleviated as the result of the intradermal *tests* before any subcutaneous therapeutic injections were given.

Phillips[32] reported the relief† of hay fever by intradermal injections of pollen extracts. He based the use of this method on the fact that tuberculin and vaccine had been given intradermally and also that hay-fever patients benefited by intradermal pollen *tests*. He reported complete relief in 2 patients desensitized intradermally, one pre-seasonally and one co-seasonally; and from 1924 he systematically employed

* The terms 'intradermal' and 'intracutaneous' appear to be used synonymously in the literature; and, in this paper, either word will be used in relation to injections *into* the skin. In the references however, the quoted author's own terminology is adhered to.

† To most writers 'relief' implies control of symptoms in the pollen season by co-seasonal therapeutic desensitization. Sometimes the reference is to pre-seasonal prophylactic desensitization before the occurrence of symptoms, and sometimes this distinction is not made clear.

the intradermal method with 2 or 3 injections a week on about half the patients treated co-seasonally. Satisfactory results, which he described as 'monotonously successful' were obtained in more than 91% of the patients, the relief being usually experienced at or before the seventh day of treatment. He stated[33] that the special advantages of this method were the low dosage required and the promptness of relief of symptoms. Duke[34] tried Phillip's intradermal technique and obtained complete relief in a remarkably short space of time in every patient treated during the season. For this reason, and because the local superficial reaction permitted a ready decision upon the size and time for a subsequent dose, he thought that this would be the method of choice. Thommen[9] used the intradermal method of desensitization with results decidedly superior to those obtained by the subcutaneous method. Similarly, Anderson[4] obtained satisfactory desensitization in 84% of his patients. Waldbott and Ascher[35] found no difference in therapeutic response from either intradermal or subcutaneous injections, but conceded that the intradermal method was of greater service as a gauge for further treatment because of the more distinct appearance of the wheal. Hansel[36] summarized reports from physicians to the effect that satisfactory relief in hay fever could be obtained by the co-seasonal method of treatment given by the intracutaneous or subcutaneous route or by both routes. He recommended that whenever possible the co-seasonal type of therapy should be preceded by a series of about 10 injections, either intracutaneously or subcutaneously, before the onset of the season. Rockwell and Rockwell[37] thought that subcutaneous injections gave good results, but had the disadvantage that the amount of reaction was not always apparent until a too severe reaction had occurred, whereas the reaction from the intradermal injection, however slight, was always visible in patients with positive skin tests.

THE USE OF THE INTRADERMAL METHOD OF DESENSITIZATION IN SOUTH AFRICA

Seasonal Pollen Allergy

Investigations into the causes and treatment of seasonal hay fever in South Africa were originally carried out by Pirie,[38] formerly of this Institute. He initially employed and advised others to use the subcutaneous method of desensitization until the intradermal method of Phillips came to his notice, when he was impressed with the results in the cases on which this was tried. He thereafter advocated the intradermal desensitization technique for pre-seasonal desensitization in hay fever and favoured the scheme of a pre-seasonal course of intradermal injections followed by injections at 7—10-day intervals throughout the season. He confirmed the advantage that Phillips had claimed for this method and added that such treatment could be started at any time either before or during the hay-fever season; that reactions were clearly visible and easily gauged, and dosage could be suitably regulated by the reaction; and that a course of treatment was usually shorter, the results better, and the risk of aggravating hay fever or of giving an anaphylactic shock by overdosage was practically nil.

More than 20 years ago when I took charge of the Allergy Department of this Institute, I had to decide whether to recommend the intradermal method or to advise the adoption of the generally used subcutaneous method. After considerable clinical and laboratory investigations I concluded that the continuance of desensitization by the intradermal route, both prophylactically and therapeutically in hay fever, was fully justified, and I have accordingly fostered this method assiduously so that it is now, with few exceptions, the method employed in South Africa.

Seasonal pollinosis in South Africa[39] is almost entirely summer hay fever or asthma caused by grass pollen present in the atmosphere from October to March. The pollens of the compositae are sometimes clinically significant more especially in florists, gardeners, and others coming into close contact with plants of this group. Not infrequently trees (mainly plane, oak, and poplar) cause symptoms when they pollinate in spring (August—October), and occasional cases of cypress (*Cupressus* spp.) pollinosis[40] occur from May to October. Ragweed (*Ambrosieae* spp.) and other weeds of importance in the United States of America play no part in respiratory allergy in South Africa.

Experience over the years has amply confirmed the effectiveness of the intradermal method of prophylactic desensitization in pollen allergy. A recent analysis of answers to a questionnaire sent to physicians in the 'grasslands' of South Africa, where summer hay fever caused by grass pollen is common, revealed undoubted benefit to the great majority of the patients. In the control of manifestations of hay fever in the season, the method employed therapeutically has proved very satisfactory since symptoms are generally alleviated after relatively few (3—12) injections of grass-pollen extract. It is wise, therefore, to encourage desensitization in the season if the patient presents himself at that time.

Non-Seasonal Allergy

The procedures of desensitization by the intradermal route have been further studied and developed to the present stage where the employment of this route can now confidently be advised for prophylaxis in any allergic condition where desensitization is indicated. Intradermal desensitization is successfully carried out in non-seasonal respiratory allergic conditions with extracts of animal hairs, feathers, house dust, air-borne fungi and the other commoner substances responsible for symptoms. Such desensitization has also been accomplished in cases of sensitivity to the inhalation of dust from wood,[41,42] cereal grains[43] and lucerne,[44] and also to the effects of bee[1,45] and wasp stings.

THE SIGNIFICANT REACTION AS A GUIDE TO DESENSITIZATION BY THE INTRADERMAL ROUTE

Desensitization by the intradermal route becomes a simple matter if the principle is followed that the occurrence of a *significant* reaction after any injection implies that desensitization is proceeding.

A *significant* reaction is represented by a well-defined, clearly visible wheal, generally with pseudopodia, 1—2 cm. in diameter appearing 5—15 minutes after an intradermal injection.

In desensitizing by the intradermal route, therefore, the aim should be to obtain a *significant* reaction after each and every injection.

With this principle as a guide it is obvious that desensitization may be commenced at any time and under any circumstances provided that the dose (size/strength) of extract producing a significant reaction has been established for the patient. Thus, in pollen sensitivity pre-seasonal desensitization may safely be followed by co-seasonal desensitization

because the dose producing the significant reaction is known at the time. For the same reason desensitization may conveniently be commenced during the pollen season. Further, if for any reason perennial desensitization with 'maintenance doses' of extracts has been interrupted (e.g. the patient has left the district temporarily or has been suffering from an unrelated illness, etc.), it may readily be resumed at any time after once again determining the dose (size/strength) of extract now required to produce a significant reaction.

RECOMMENDED METHOD OF DESENSITIZATION BY THE INTRADERMAL ROUTE

Outline of Procedures

The desensitization procedures by the intradermal method involve:

(*a*) The determination of the initial dose of the extracts to be employed, i.e. the smallest dose (size/strength) required to produce a *significant* reaction on intradermal injection.

(*b*) Systematic desensitization thereafter with gradually increasing doses until the full strength (1 : 1) extract is reached, aiming with each injection to obtain a *significant* reaction.

(*c*) Continuing with 'maintenance' injections of the full strength (1 : 1) extract at increasing intervals of time to maintain the state of hyposensitization acquired.

Details of Techniques and Procedures

1. *Preparation of Extracts*

Watery extracts of pollens and other allergens are prepared in the usual way with buffered saline solutions (Coca's, Evan's, etc.). Stock extracts are prepared according to the weight/volume relationship of allergen to extracting fluid. These are for convenience referred to as 'full strength' (1 : 1) extracts from which dilutions are made as required for intradermal desensitization. Determinations of protein nitrogen or other 'unitage' are not made.

2. *Treatment Sets for Intradermal Desensitization*

Treatment sets for desensitization purposes are, for convenience, prepared for each patient in a series of 5 graduated standard strengths of extracts in 2 ml. rubber-capped bottles:

1 : 1,000, 1 : 100, 1 : 10, 1 : 2, 1 : 1.

If the patient is previously known to be highly sensitive (clinically) to a specific substance, the treatment set may consist of a series of extracts weaker than the above, commencing, for example, with 1 : 10,000, 1 : 100,000, or even greater dilutions.

3. *Determination of the Initial Dose*

The dose (size/strength) for the initial injection will depend upon the degree of the patient's sensitivity to the graduated extracts in the set, and is readily determined by a series of intradermal injections of 0·05, 0·1 and 0·2 ml. of each strength of extract, commencing with the weakest at intervals of 5—15 minutes until a *significant* reaction appears. In most patients the significant reaction appears with the 1 : 100 strength, although the occasionally more highly-sensitive persons may react to the 1 : 1,000 strength extract.

4. *Desensitizing Injections*

The volume of an intradermal injection does not exceed 0·25 ml. A fresh area of skin is selected for each injection to ensure a brisk local response and the next injection should be given not sooner than 24 hours thereafter by which time the previous reaction will have faded.

Injections for desensitization are given daily or every other day with the weaker extracts (1 : 1,000 or 1 : 100) and 2 or 3 times a week with the stronger extracts (1 : 10, 1 : 2, 1 : 1).

The aim with each and every injection is to obtain a wheal 1—2 cm. in diameter. As long as that occurs desensitization is proceeding and there is no need to increase size of dose or strength of extract, even though the same dose (size/strength) is repeated a number of times. When, however, the reaction wheal tends to become smaller, the dose-size or extract-strength or both are systematically increased so that the required significant reaction constantly appears.

The number of injections that need to be systematically given is indicated in Table I, but will of course depend on the way the patient has reacted. Sometimes certain doses of particular size/strength may have to be repeated a number of times because of the persistence of a significant reaction after each injection. This should occasion no concern because

TABLE I. OUTLINE OF DESENSITIZATION BY THE INTRADERMAL ROUTE

Strength of extract	*Size of injection ml.*	*Comments*
1 : 1,000	0·05 0·10 0·20	The initial dose for desensitization, as indicated by the *significant* reaction, is generally within this range
1 : 100	0·05 0·10 0·15 *** 0·20 ***	
1 : 10	0·05 *** 0·10 *** 0·15 *** 0·20 ***	
1 : 2	0·05 *** 0·10 *** 0·15 *** 0·20	***Any of these doses may have to be repeated one or more times depending on the persistence of the *significant* reaction
1 : 1	0·10 *** 0·15 *** 0·20 ***	
	'Maintenance' doses	Injections at gradually increasing intervals until an injection is given every 6—10 weeks

desensitization is occurring; and often in such cases the maintenance series of injections may be commenced even before the full-strength extract is reached, i.e. with the 1 : 2 or even occasionally with the 1 : 10 strength extract. But with each and every such injection it is important that a significant reaction be obtained.

5. Maintenance Injections

When the patient readily tolerates the full-strength (1 : 1) extract, i.e. responds to it with a significant reaction, the intervals between injections are increased until he is receiving an intradermal injection once a week for a few weeks, once a month for 2 or 3 months, and thereafter once every 6 - 10 weeks as a 'maintenance' dose to preserve the state of hyposensitization acquired. Indeed the physician will often be able to space the maintenance injections at even longer intervals depending upon the continued well-being of the patient. It is necessary, however, to ensure that a significant reaction is consistently obtained after each and every such maintenance injection. With this use of maintenance injections in pollen allergy the need for subsequent pre-seasonal desensitization injections is obviated.

Reactions

If the principle of aiming for a significant reaction is followed there is no need to anticipate any general or even undue local reactions.

The objections reported to the use of the intradermal route for desensitization have no validity with the techniques recommended and employed by us. Duke[46] and Vaughan and Black[47] referred to 'tissue-scarring' as a complication. Phillips[33] had already in 1933 commented on skin blemishes after intradermal injections and showed that these were from scratching or the intolerance of certain skins to glycerine, which was sometimes used at that time with desensitizing extracts or for the incorporation therein of adrenaline. In our experience scarring never occurs with the use of the usual buffered saline extracts of allergens. Thommen[9] and Rockwell and Rockwell[37] pointed out that the limited amount of extract that could be injected was a disadvantage. The implication in this adverse comment is that relatively large amounts of extracts are necessary for desensitization purposes. In our experience of intradermal desensitization the important matter is not the quantity of extract in each dose or in the total amount injected, but as has been shown, in the production after each injection of a *significant* skin reaction; and for this small amounts only of extract are, in fact, required. This view is supported by Phillips,[33] who had previously stated that the relief was proportionate to the extent and vigour of the local reaction rather than to the quantity of pollen extract administered, as well as by Anderson[4] who emphasized that with the intradermal technique small amounts of pollen extracts were adequate in the treatment of the great majority of patients. Hansel[36] also reported that small amounts only of the extract proved effective, the degree and duration of relief being generally proportional to the size of the wheal as well as to the amount and strength of the dilutions employed.

SUMMARY AND CONCLUSIONS

While the subcutaneous route is almost generally employed for desensitization in allergic conditions, the intradermal method is and has been the method mainly used in South Africa—its history and development are described.

Details are given of the principles, procedures, and techniques at present recommended by us for intradermal desensitization.

Emphasis is placed on the importance of following the principle: that desensitization is occurring if a *significant* reaction follows an intradermal injection and on the necessity, therefore, of always aiming for a *significant* reaction—a wheal 1—2 cm. in diameter, generally with pseudopodia.

Advantages claimed for the intradermal method of desensitization include the following:

(*a*) The superficial local skin reactions after intradermal injections are clearly visible within a few minutes after the injection and provide an adequate guide to the dose (size/strength) of the injection to follow.

(*b*) With the *significant* reaction as a guide, desensitization may be commenced or resumed at any time and under all circumstances, irrespective of season or other contact with the responsible allergen.

(*c*) The amount of extract used for each injection and for the desensitization procedure as a whole is relatively small.

The intradermal method is recommended because of its effectiveness in pre-seasonal and co-seasonal desensitization in pollen allergy and also for its value in prophylactic desensitization with extracts of other allergens responsible for non-seasonal allergic conditions.

With the procedures recommended, this method is noteworthy for the absence of severe local and any general reaction.

A successful outcome from intradermal desensitization may be anticipated where the specific allergens responsible for the condition are clearly defined and used for desensitization and where the recommended procedures are followed.

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EDITORIAL : VAN DIE REDAKSIE

CRYPTOCOCCOSIS

Cryptococcosis can no longer be regarded as a laboratory curiosity; it is neither rare nor uniformly fatal. In the United States of America, approximately 10% of the 300 - 400 annual deaths from mycotic infections are cryptococcal in origin.[1] At Groote Schuur Hospital, Cape Town 19 cases have been collected, 15 of which have occurred during the last 4 - 5 years.[2,3]

The causal organism is a simple fungus, which reproduces by budding, and is unique in that it produces a mucinous capsule in tissue and in culture—this is shown up well on a wet Indian-ink preparation. No mycelia or ascospores are formed. Growth occurs aerobically on Sabouroud's medium, but this may take up to four weeks. Pathogenicity is proved by intracranial inoculation into suckling mice.

Cryptococcus neoformans has been isolated from grasses, insects, pigeon excreta, bees' nests, soil, and the throat, skin, and gastro-intestinal tract of normal healthy people. What renders a particular strain pathogenic is not known. Physical factors or antigenic structure may play a part. Infection has never been shown to be transmitted from man to man, or animal to man,[4] though pigeons are suspect.[5] The mode of infection in man is likewise a mystery; probably by inhalation of air-borne fungi, with subsequent haematogenous dissemination, the lungs being the primary portal of entry. However, recent work suggests that the gastro-intestinal tract may also play a part.[6]

Clinically, the disease is protean. It may present as a pulmonary infiltration, single or multifocal, and may thus simulate carcinoma, tuberculosis, and sarcoid. Dissemination occurs most frequently to the brain and meninges where the parasite finds the concentration of thiamine and glutamic acid optimum for growth.[7] The clinical features are those of a chronic meningitis, with changes in the cerebrospinal fluid which may resemble tuberculous infection. In more chronic cases, cryptococcosis takes the form of a large, single, intracerebral granuloma—and thus presents as a space-occupying mass. Skin (acneform, granulomatous or ulcerative lesions), bone and joint, ocular, renal, and adrenal involvement have been reported. In all cases, the diagnosis depends on the isolation of the organism and proof of its pathogenicity. The coexistence of this condition with chronic debilitating disease, especially of the reticulo-endothelial system, e.g. lymphomata, is noteworthy and this possibility should be borne in mind when a patient with a chronic disease deteriorates.

It has been pointed out that cryptococcosis is no longer uniformly fatal. Treatment of the condition has, until recently, been unrewarding. Antibiotics and sulphonamides have no effect, and steroids actually cause a flare-up in laboratory animals. Actidione (cyclohexamide), an antibiotic from *Streptomyces griseus*, has proved disappointing in man after promising *in vitro* tests.[8-11] Polymyxin B, nystatin, and 2 hydroxystilbamidine have not altered the fatal outcome.

A drug of distinct promise is amphotericin B, an antibiotic derived from a species of South American streptomyces. It has not only been found extremely effective *in vitro*[12] and in laboratory animals,[13,14] but preliminary reports have also confirmed its efficacy in human infection. A review of the current literature [5,15-22] reveals a mortality in cryptococcal meningitis vastly different from the previous figure of 100%. While all patients have not been followed up for long enough periods (three months to two years in most instances) a significant number of patients have recovered completely, with a return of the cerebrospinal fluid to complete chemical and bacteriological normality. A mortality of 30% in the 50 cases reviewed is noted.

Amphotericin is, however, not free from side-effects, which include troublesome rigors and occasional toxic reactions in the kidney and bone marrow. Moreover, it has to be infused intravenously over long periods.

Littman,[5] who has shown thiamine to be an essential requirement for good capsule formation by the organism, suggests a low thiamine diet in addition, but this seems rather drastic in an already ill and depleted patient. Finally, since many of his more recent patients have had a low serum gamma-globulin fraction, Littman recommends gamma-globulin supplements bi-weekly.

Reasonably effective therapy for what was formerly a fatal illness makes early recognition of cryptococcosis imperative. It is clear that doctors must regard unusual pulmonary and neurological syndromes with a higher index of suspicion. Into this category, too, must fall such entities as non-healing ulcers or obscure skin granulomata, atypical osteitis[23], unexplained pyrexias and generalized adenopathy.

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MOTORKOMPETISIE TEN BEHOEWE VAN DIE LIEFDADIGHEIDSFONDS

As gevolg van die inisiatief van mev. E. Getaz, 'n lid van die Vereniging van Mediese Vrouens van die Tak Kus, Natal, van die Mediese Vereniging, het die geleentheid nou ontstaan om 'n aansienlike bedrag bymekaar te maak vir die Liefdadigheidsfonds van die Mediese Vereniging. Die Standard Triumph (Edms.) Bpk. Maatskappy, het 'n Triumph Herald koepee motor geskenk as 'n prys in 'n kompetisie. Die kompetisie word op 'n nasionale basis georganiseer deur die advertensiefirma Lindsay Smithers (Edms.) Bpk. Die opbrengs van die verkoop van kaartjies, sonder enige aftrekkings, hoegenaamd, sal na die Liefdadigheidsfonds gaan.

Dit is 'n baie groot geleentheid vir die Liefdadigheidsfonds, wat dit maar altyd baie moeilik vind om aan die baie dringende en dikwels tragiese beroepe te voldoen wat op hom gemaak word, om R20,000 of meer te kry. 'n Minimum van 75,000 kaartjies wat teen 25c elk verkoop sal word, sal uitgereik word. Die verwagting is dat alle lede van die Mediese Vereniging sal saamwerk om die kaartjies te koop en te verkoop. 'n Hele aantal Takke het alreeds getoon dat hulle besonder baie in die projek belang stel. Die Tak Kus, van Natal, waar die gedagte ontstaan het, sal natuurlik baie kaartjies verkoop, en die Tak Suid-Transvaal het 40,000 kaartjies aangevra om in Johannesburg en die omliggende gebied te verkoop. Die Tak Wes-Kaapland hoop ook om ten minste 20,000 kaartjies te verkoop. Besonderhede van die reëls verskyn op die inskrywingsvorms wat saam met elke kaartjie wat verkoop word, uitgegee word. Die hoop word gekoester dat die motor by die geleentheid van die 43ste Mediese Kongres in Kaapstad in September aan die wenner van die kompetisie oorhandig sal word. Die kaartjies sal verkoop word gedurende die maande Julie en Augustus.

Dr. E. W. Turton, voorsitter van die Federale Raad, stem saam dat hierdie plan, alhoewel dit iets nuuts is wat betref geldinsameling vir die Liefdadigheidsfonds, 'n baie goeie manier is om die inkomste van die Fonds te vergroot. Hy doen dan ook 'n beroep op alle lede van die Vereniging en hul vrouens om alles in hulle vermoë te doen om soveel kaartjies as moontlik te verkoop.

Dit is miskien juis die vrouens van lede wat die grootste bydrae sal kan maak om die projek te laat slaag. Dameskomitees is alreeds gevorm of is besig om gevorm te word in die grotere sentrums van die land, en alle vrouens van dokters op wie nog nie 'n beroep om hulp gedoen is nie, word ernstig versoek om dienste aan te bied in hierdie verband.

Sommige lede van die Vereniging mag wel bedenkings hê oor hierdie metode van geldinsameling vir die Liefdadigheidsfonds. As die volgende belangrike feite egter in gedagte gehou word, sal die saak in sy regte perspektief gesien kan word.

Om mee te begin, het die Liefdadigheidsfonds geld baie nodig. Ons weet almal maar te goed hoe baie dokters in die loop van hul beste jare sterf, voordat hulle in staat was om geskikte voorsiening te maak vir hulle afhanklikes.

Dit is ontstellend om die navrae en die eisevorms wat die bestuurskomitee van die Liefdadigheidsfonds ontvang, na te gaan. Ook is dit onstellend dat so baie van die aansoeke om hulp van die hand gewys moet word of dat slegs 'n paar rand per maand toegestaan kan word terwyl veel meer nodig is. Die ou opvatting dat alle dokters ryk sterf, berus op heeltemal valse gronde. Tot nog maar onlangs was daar net 'n paar maniere waarop die Fonds sy kapitaal kon vergroot. Hierdie maniere het bestaan uit *In memoriam* bydraes vir dienste wat gelewer is, nalatings deur dokters, en donasies. Gedurende die laaste aantal jare het verskeie Takke spesiale funksies gehou, soos danse en bioskoopvertonings wat taamlike groot somme geld vir die Fonds opgelewer het. Hierdie pogings, verdienstelik soos hulle is, is egter nie voldoende vir al die eise wat daar aan die Fonds gestel word nie.

Die huidige kompetisie kan 'n groot bydrae maak wat betref die hulp vir afhanklikes van lede wat dringende finansiële ondersteuning nodig het. Dit is waarskynlik waar om te sê dat dokters, meer as die lede van enige ander professie, hulle dienste vry beskikbaar stel vir lede van die samelewing. Dit is goed dat die mediese professie ingestel is daarop om te dien, en die groot hoeveelheid moeilike, onbetaalde, en erewerk wat deur die meeste van ons kollegas gedoen word, word wel met dankbaarheid deur hul pasiënte onthou. 'n Groot aantal *In memoriam* bydraes word dan ook deur die Fonds ontvang deur dankbare pasiënte wat bekend is met die opofferings wat individuele dokters gedurende hul leeftyd gemaak het. Ons is oortuig daarvan dat die reaksie van die publiek ten opsigte van die verkoop van hierdie kaartjies oorweldigend sal wees, omdat hulle weet dat al die geld gebruik sal word vir die Liefdadigheidsfonds.

Om die redes wat ons hier genoem het, voel ons seker dat almal heelhartig sal bydra om van hierdie onderneming 'n groot sukses te maak. Elke Tak en Afdeling van die Vereniging sal informasie ontvang aangaande die kompetisie, insluitende kennisgewings en besonderhede in verband met die reëls van die kompetisie. Hierdie informasie kan dan in spreekkamers, hospitale, en verpleeginrigtings opgeplak word. Ons is seker daarvan dat lede en hulle vrouens sal toesien dat geen kaartjie onverkoop sal oorbly nie, maar dat dit selfs nodig sal wees om nog meer kaartjies te laat druk. Op hierdie manier kan 'n wonderlike gebaar ten behoewe van die Liefdadigheidsfonds 'n groot sukses word.

THE SIGNIFICANCE OF MECONIUM IN FOETAL DISTRESS

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When pregnancy ends with the delivery of an infant that is either dead, dying, or destined to be mentally defective, it is indeed a tragedy. On recognizing the signs of foetal distress, the accoucheur can frequently prevent this unfortunate outcome by timely intervention. No greater test of the obstetrician's judgment and clinical acumen exists, for the wrong decision can too frequently end in disaster.

The criteria for the diagnosis of foetal distress are well known. Various authors have attempted either to minimize or to emphasize the importance and value of certain signs

as indications of foetal distress.[1-4,6,9-11,14-16,18,19,21-27] Of all the signs, the significance of meconium in the liquor amnii associated with vertex presentations is probably the most controversial.[8,13,17,21,23-26]

In this paper I propose (*i*) to assess whether the recognition of meconium in the liquor amnii in vertex presentations is sufficiently serious to warrant interference, either immediately or at a later stage; (*ii*) to show what the outcome was in cases where conservatism was practised; and (*iii*) to suggest a scheme of treatment when meconium is recognized during the course of labour.

METHODS OF STUDY

A study was made of 2,600 consecutive deliveries at the Somerset Hospital, Cape Town, between 1 January 1957 and 31 May 1958. These patients were drawn from the non-White population (mainly from Cape Town). The proportion of Coloured to Bantu admissions was approximately 2½ : 1 (1,863 Coloured and 730 Bantu patients). Seven Asian women were delivered in hospital during this period. These patients are a selected group, since only primigravidae, grande-multiparae (over 5 pregnancies), and patients with a poor obstetrical history or with antenatal complications are admitted to the Somerset Hospital for indoor delivery.

In all vertex deliveries the presence of meconium was noted as a routine by the nursing staff. The degree of meconium (light or frank), and the colour (light-green or dark olive-green) was recorded. A more frequent and strict checking of the foetal heart (¼-hourly whenever possible) was started immediately meconium was seen. Unless the delivery was imminent, a member of the medical staff was always informed and a vaginal examination done. In order to check the records of the nursing staff, I personally recorded 44 random cases. Subsequent comparison indicated that they had not failed to observe the presence of meconium in any of these cases. Unless confirmed by the medical staff, a single observation of 'slightly meconium-stained liquor' was not accepted for this series.

INCIDENCE

Meconium was present in association with vertex deliveries in 169 of the 2,600 cases studied. Bearing in mind the selected nature of the patients, this incidence of 6·5% cannot be accepted as representative of all cases. Other authors have quoted figures varying from 3% to 9·6%.[5,21,26] In the 169 cases studied, there were 93 primigravidae and 76 multigravidae, whereas in the overall admissions there was a higher incidence of multigravidae. Patients with the 2 types of meconium, namely, 'light meconium' and 'frank or dark-green meconium' are referred to throughout the rest of the paper as 'group 1' and 'group 2' respectively. There were 120 patients in group 1 and 49 in group 2.

Perinatal Mortality

Altogether 23 infants were lost during labour and the first week of the puerperium. This represents an incidence of 13·6%. Since these included 8 'dead births' (the foetal hearts not being heard on admission or before the onset of labour), the salvable foetal loss was 9·3%.

The uncorrected perinatal mortality found by other authors was 5·3%[17] and 8%.[12] Besides the 8 babies who were not salvable, there were 10 stillbirths and 5 neonatal deaths. In group 1 (120 cases) 6 infants were lost, an incidence of 5%. The salvable incidence was 3·4%. In group 2 (49 cases) 17 infants were lost, an incidence of 34·7%. The percentage of babies that could have been saved in this group was 25·6% (Table I). *The marked difference in foetal loss in group 2 compared with group 1 serves to emphasize the importance that should be attached to the presence of frank meconium.* This view is confirmed in other publications,[7,8,21] but Walker[25] found a higher loss associated with thin meconium (11·1%) than with thick meconium (7·8%).

Perinatal Mortality in Relation to Method of Delivery

Spontaneous vaginal delivery occurred in 133 cases, an incidence of 78·7%. There were 21 forceps deliveries (12·4%) and 15 Caesarean sections (8·9%).

(i) *Spontaneous vaginal delivery* (*Table II*). The overall loss for this group was 7·1%, while the loss associated with group 2 was 8 times that of group 1. Of the 3 neonatal deaths in group 1, 2 could have been prevented by earlier intervention. Both mothers had premature rupture of the membranes. One of the cases was associated with toxaemia and postmaturity, the other with a mild accidental haemorrhage and prematurity. Of the 6 salvable babies in group 2, earlier intervention was indicated in 2 cases and could have saved both infants. One case was associated with toxaemia, and thick meconium appeared some time after rupture of the membranes. The cord was found to be wound tightly round the neck at birth. The other case was associated with postmaturity and a mild accidental haemorrhage. Meconium was noticed when the os was 2 fingers dilated. In both cases, no other signs of foetal distress were present and consequently intervention was not considered. Of the 9 babies lost in spontaneous vaginal delivery, it would thus appear that 4 could have been saved by early intervention. In the 5 remaining cases, meconium was noticed too late for intervention to have altered the outcome.

TABLE I. PERINATAL MORTALITY IN RELATION TO PARITY

	Parity	*No. of cases*	*SB*	*DB*	*NND*	*No. lost*	*Uncorrected loss %*	*Salvable loss %*
Group 1	P	69	—	1	1	2	2·9	1·5
	M	51	1	1	2	4	7·8	6·0
	Total	120	1	2	3	6	5·0	3·4
Group 2	P	24	4	4	1	9	37·5	25·0
	M	25	5	2	1	8	32·0	26·1
	Total	49	9	6	2	17	34·7	25·6
Total		169	10	8	5	23	13·6	9·3

SB=stillbirth, DB=dead birth, NND=neonatal death, P=primipara, M=multipara.

(ii) *Forceps delivery* (*Table III*). The overall loss after forceps delivery was 10%. In group 1, forceps were applied for foetal distress *per se* on only 7 occasions; an irregular foetal heart was present each time. Earlier intervention could have prevented the 1 stillbirth, since meconium appeared 15 hours before delivery, the foetal heart becoming faint and irregular 7 hours later. At birth the cord was found to be wound tightly round the neck. In group 2, forceps were applied primarily for foetal distress on 4 occasions; all were associated with an irregular foetal heart rate. The stillbirth in this group could not have been prevented, since the foetal heart sounds disappeared without earlier irregularity 18 hours before delivery, meconium being noticed for the first time 5 hours later. The cord was once again found to be wound tightly round the neck at birth. Of the 2 stillbirths in this section, only 1 could have been prevented by early intervention.

TABLE II. PERINATAL LOSS ASSOCIATED WITH SPONTANEOUS VAGINAL DELIVERY

	Parity	Cases	SB	DB	NND	Corrected loss %
Group 1	P	58	—	1	1	1·8
	M	45	—	1	2	4·6
	Total	103	—	2	3	3·0
Group 2	P	15	1	3	—	8·3
	M	15	4	2	1	38·5
	Total	30	5	5	1	24·0
Total—groups 1 and 2		133	5	7	4	7·1

For key, see Table I.

(iii) *Caesarean section* (*Table IV*). The percentage perinatal loss after Caesarean section was almost 3 times that of the series as a whole (26·7% compared with 9·3%). This might lead to the fallacious conclusion that Caesarean section should be employed less frequently. However, many Caesarean sections were undertaken for indications other than foetal distress. In group 1, only 3 sections were performed primarily for foetal distress. All 3 babies were born alive. In group 2, 4 out of the 10 sections were performed primarily for foetal distress; all 4 of these infants survived. In 4 cases where the section was performed for disproportion, there were 2 stillbirths. In both of these meconium appeared long before the sections were done and earlier operation could have saved both these babies. From the above analysis, it is seen that, in the 7 cases where a Caesarean section was undertaken for foetal distress, no infant was lost. If heed had been taken of the frank meconium at an earlier stage, 2 more infants might have survived.

TABLE III. PERINATAL LOSS ASSOCIATED WITH FORCEPS DELIVERY

	Parity	Cases	SB	DB	NND	Corrected loss %
Group 1	P	7	—	—	—	—
	M	5	1	—	—	20·0
	Total	12	1	—	—	8·3
Group 2	P	5	1	1	—	25·0
	M	4	—	—	—	—
	Total	9	1	1	—	12·5
Total—groups 1 and 2		21	2	1	—	10·0

For key, see Table I.

FOETAL LOSS IN RELATION TO THE TIME OF FIRST APPEARANCE OF MECONIUM BEFORE DELIVERY

Table V shows that 10 out of the 15 salvable foetal deaths occurred when meconium was present for 6 or more hours before delivery. Two further Caesarean sections were performed in primigravidae for indications other than foetal distress. The meconium was noticed for the first time at operation (dilatation less than 3 fingers in both cases) and it can be presumed that delivery might have taken 6 or more hours had a vaginal delivery been possible. The 1 multigravida grouped in the section 'under ½-hour' was admitted with the foetal head on the perineum, and should thus correctly not be grouped in this section, meconium having been present for an undetermined length of time before admission.

OUTCOME IN RELATION TO DILATATION OF CERVICAL OS

For the purpose of investigating the outcome in relation to cervical dilatation, the patients were divided into 3 sections:

1. Those able to be delivered immediately (i.e. os either fully dilated or almost fully dilated).
2. Those where the os was dilated 3 fingers or less.
3. Those where the os was dilated more than 3 fingers, but was not near full dilatation.

Section 1 (*67 patients*)

Fifty-eight patients were delivered spontaneously per vaginam (Table VI). The remaining 9 were delivered by forceps. Of these, forceps were applied in 5 for foetal distress *per se*. Of the 8 infants lost, only 2 were salvable, a corrected foetal loss of 3·3%. In neither of these patients did meconium appear early enough to indicate intervention. The 5 infants delivered by forceps might well have survived because of the presence of meconium, for it was this sign which drew attention to the fact that something was amiss and called for a closer watch for alterations in the foetal heart rate.

TABLE IV. PERINATAL LOSS ASSOCIATED WITH CAESAREAN SECTION

	Parity	Cases	SB	DB	NND	Corrected loss %
Group 1	P	4	—	—	—	—
	M	1	—	—	—	—
	Total	5	—	—	—	—
Group 2	P	4	2	—	1	75·0
	M	6	1	—	—	16·7
	Total	10	3	—	1	40·0
Total—groups 1 and 2		15	3	—	1	26·7

For key, see Table I.

Section 2 (*58 patients*)

The foetal loss associated with frank meconium (35·3%) was approximately 7 times as high as that associated with thin meconium (5·1%)—Table VII. Only 5 of the 11 Caesarean sections performed here were primarily for foetal distress. None of these infants was lost. Had a Caesarean section been done earlier in the following case, the baby might have been saved:

A 26-year-old grav. ii, para. i, 40 weeks pregnant by dates, was in labour for 36½ hours. Frank meconium presented with rupture of the membranes 19½ hours before a Caesarean section was performed at 3 fingers dilatation. The foetal heart sounds became irregular 5 hours before and disappeared altogether 3 hours before the Caesarean section, which was eventually undertaken for obstructed labour. The birth weight of the baby was 9 lb. 7 oz.

In the spontaneous vaginal births in this section, 4 of the 5 infants lost could have been saved by early intervention:

(i) A 24-year-old primigravida, 42½ weeks pregnant by dates, with mild toxaemia, was in labour for 37 hours. The membranes ruptured 72 hours before delivery, meconium being present 8 hours before the birth. No antibiotics were given until admission 24 hours before delivery. The baby died after 13 hours. A postmortem examination showed signs of aspiration pneumonia.

(ii) A 22-year-old grav. ii, para. i was at term. The foetal heart sounds disappeared suddenly 20 hours before delivery, after the patient was in labour for 21 hours. When the membranes

TABLE V. FOETAL LOSS IN RELATION TO TIME MECONIUM FIRST NOTICED BEFORE DELIVERY

Group	Parity	Under ½ hour	½ - 1 hr.	1 - 1½ hrs.	1½ - 3 hrs.	3 - 6 hrs.	6 - 12 hrs.	Over 12 hrs.
1	P	—	—	—	—	—	—	1
	M	—	—	—	—	1	1	1
2	P	1	—	—	—	—	1	1
	M	1	—	—	—	—	2	3
1 + 2	Total	2	—	—	—	1	4	6

2 infants were born by Caesarean section, meconium being noticed for first time at section.

TABLE VI. PATIENTS ABLE TO BE DELIVERED IMMEDIATELY. METHOD OF DELIVERY AND PERINATAL MORTALITY

Group	Parity	No. of cases	NVD	LF	MF	HF	CS	SB	DB	NND	Corrected loss %
1	P	29	26	2	1	—	—	—	—	—	—
	M	14	13	1	—	—	—	—	1	—	—
	Total	43	39	3	1	—	—	—	1	—	—
2	P	13	11	1	1	—	—	1	3	—	10
	M	11	8	1	2	—	—	—	2	1	11·1
	Total	24	19	2	3	—	—	1	5	1	10·5
1 + 2	Total	67	58	5	4	—	—	1	6	1	3·3

NVD = normal vaginal delivery, LF = low forceps, MF = mid forceps, HF = high forceps, CS = Caesarean section, SB = stillbirth, DB = dead birth, NND = neonatal death, P = primigravida, M = multigravida.

TABLE VII. DILATATION OF OS 3 FINGERS OR LESS (58 CASES). METHOD OF DELIVERY AND PERINATAL MORTALITY

Group	Parity	No. of cases	NVD	LF	MF	HF	CS	SB	DB	NND	Corrected loss %
1	P	21	14	2	1	1	3	—	1	1	5·0
	M	19	17	—	2	—	—	1	—	—	5·7
	Total	40	31	2	3	1	3	1	1	1	5·1
2	P	6	3	1	—	—	2	1	1	—	16·7
	M	12	6	—	—	—	6	5	—	—	41·7
	Total	18	9	1	—	—	8	6	1	—	35·3
1 + 2	Total	58	40	3	3	1	11	7	2	1	14·3

For key, see Table VI.

TABLE VIII. OS OVER 3 FINGERS DILATED, NOT FULLY DILATED. METHOD OF DELIVERY AND PERINATAL MORTALITY

Group	Parity	No. of cases	NVD	LF	MF	HF	CS	SB	DB	NND	Corrected loss %
1	P	19	18	—	—	—	1	—	—	—	Nil
	M	18	15	—	1	1	1	—	—	2	11·1
	Total	37	33	—	1	1	2	—	—	2	5·4
2	P	5	1	2	—	—	2	2	—	1	60·0
	M	2	1	—	1	—	—	—	—	—	Nil
	Total	7	2	2	1	—	2	2	—	1	42·8
1 + 2	Total	44	35	2	2	1	4	2	—	3	11·3

For key, see Table VI.

ruptured half an hour later, thick meconium was present. The placenta was grossly infarcted. (Had the membranes been ruptured earlier, the presence of meconium might have called for intervention and the infant might have been saved.)

(iii) A 45-year-old grav. ix, para. vii, 41 weeks pregnant by dates, was in labour for 63 hours. Meconium appeared 6 hours before delivery. The foetal heart rate was regular, but the sounds disappeared suddenly 30 minutes before the birth. The cord was wound tightly round the neck.

(iv) A 42-year-old grav. xv, para. xiv, 42 weeks pregnant by dates, was admitted to hospital with a mild accidental haemorrhage. Thick meconium was present with the rupture of the membranes 8½ hours before delivery. The foetal heart rate was regular, but the sounds suddenly disappeared 2½ hours after the membranes ruptured.

Section 3 (44 patients)

The number of patients in group 2 in this section is too small for statistical evaluation, but the overall mortality of 42·8% for this group, compared with 5·4% for group 1, once again illustrates the increased foetal loss associated with the presence of frank meconium (Table VIII). Only 1 of the 5 forceps deliveries in this section was done for foetal distress. Of the 4 Caesarean sections, only one was performed because of the presence of meconium—a grande-multipara with no live children. The baby was born alive. Of the 5 infants lost in this section, meconium appeared too late for intervention in 3 cases, 1 infant had a meningocele incompatible with life, and the fifth infant might have been saved by earlier intervention:

A 35-year-old grav. viii, para. vii, 34 weeks pregnant by dates, was admitted in labour with a mild accidental haemorrhage. Light meconium was noticed 4 hours after the onset of labour, the foetal heart being slightly irregular. The infant was born limp 9 hours later, responding to resuscitation after 30 minutes. Its birth weight was 3 lb. 13 oz., and it died after 5 hours.

CONGENITAL ABNORMALITIES

There were only 4 infants with congenital abnormalities. One was insignificant (extra digits only). The 3 remaining infants were born dead. In 2 of these foetal heart sounds were not heard on admission, the babies being *(i)* a monster with numerous abnormalities and *(ii)* a hydrocephalic. The third baby, which was stillborn, had a meningocele incompatible with life.

The peculiarly low incidence of congenital abnormalities in this series (2·4%) is interesting in view of the suggestion made by Leslie[12] that a considerable chance of finding a congenital abnormality existed (the incidence in his series was 6·4%). A conservative attitude with regard to Caesarean section because of the fear of delivering a congenitally abnormal child is not substantiated by the present series.

MANAGEMENT OF CASES WITH MECONIUM

A retrospective analysis of the present series indicated that vertex deliveries associated with meconium should be managed along the following lines:

Once meconium is present, an immediate vaginal examination must be carried out to exclude a prolapse or 'nipping' of the cord. The dilatation of the os is now of major importance and serves as a guide in anticipating roughly how much longer it will be before the infant can be born. The foetal heart must be checked and recorded regularly at quarter-hour intervals.

The case is now assessed, paying particular attention to the following points: *(i)* Are there any other signs of foetal distress? *(ii)* Are any of the following associated factors present—toxaemia, postmaturity, disproportion, accidental haemorrhage, elderly primiparity, prolonged labour? *(iii)* What sort of meconium is present—thin and light-green, or thick, frank, and dark-green? The cases are then divided into 2 groups—*(a)* light meconium and *(b)* frank meconium.

Group 1 (Light Meconium)

(i) *Three Fingers or less Dilatation*

(a) No other sign of foetal distress and no associated factors present. These patients generally have a good prognosis and can be left alone while checking the foetal heart carefully.

(b) Other signs of foetal distress are present—a Caesarean section should be done.

(c) In the presence of the associated factors mentioned above, no intervention is recommended at this stage, purely because of the appearance of thin meconium. If, however, any obstetrical abnormalities may require interference at a later stage, then immediate intervention is indicated. *No further trial of labour* should be allowed. If a Caesarean section is not indicated, a forceps delivery should be carried out at full dilatation.

(ii) *Over 3 Fingers Dilated, but not near Full Dilatation*

(a) No other signs of foetal distress and no associated factors present—leave alone while checking the foetal heart carefully.

(b) If other signs of foetal distress are present, a Caesarean section must be done.

(c) Should associated factors be present, conservatism must be tried, with, however, a more frequent reassessment of the case. An unsatisfactory progress of the labour requires a Caesarean section sooner than would normally be necessary in the absence of meconium. Forceps delivery should be carried out at full dilatation.

(iii) *Full Dilatation or Almost Fully Dilated*

(a) If there are no other signs of foetal distress or any associated factors, spontaneous delivery is allowed.

(b) In the presence of other signs of foetal distress or associated factors, a forceps delivery should be carried out as soon as practicable.

Group 2 (Frank Meconium)

(i) *Three Fingers or less Dilatation*

Bearing in mind the high foetal mortality associated with this group, Caesarean section is recommended, *even in the absence of other signs of foetal distress or associated factors.*

(ii) *Over 3 Fingers Dilated, but not near Full Dilatation*

(a) In the absence of any other signs of foetal distress or associated factors, a conservative attitude is justifiable, provided a Caesarean section can be performed at short notice. Delivery by forceps at full dilatation is essential.

(b) In the presence of other signs of foetal distress or any of the associated factors, a Caesarean section should be performed.

(iii) *Full Dilatation or Almost Fully Dilated*

Delivery by forceps is clearly indicated, irrespective of whether additional signs of foetal distress or other associated factors are present.

In addition, it is suggested that a high rupture of the membranes be performed as a diagnostic procedure whenever a trial of labour is anticipated under the following conditions: elderly primipara, postmaturity or toxaemia.

Likewise, in prolonged labour, if the membranes are intact, a diagnostic rupture should be done.

Although oxygen was administered to most of the mothers in this series, its value at the present time is still debatable and does not alter the scheme of treatment in any way.

DISCUSSION

Of all the signs of foetal distress, the significance of meconium in the liquor amnii in vertex presentations is probably the most controversial. In the literature it is generally considered to be associated with a favourable outcome in the absence of any other signs of foetal distress, serving merely as a warning of impending danger. As such it calls for a close watch on the foetal heart. There is general agreement that, if associated with other signs of foetal distress, its significance is increased considerably and the need for intervention seems indicated.[5,10,12,17,20,26]

The present investigation led to the following conclusions: The presence of frank, dark-green meconium was far more significant than that of thin, light-green meconium, the foetal loss in the present series being nearly 8 times as high when associated with frank meconium (25·6%) as compared with thin meconium (3·4%). Other authors report similar findings.[8,13] In elderly primigravidae and in postmaturity, meconium is a serious sign.[21,23,24] In such cases the foetal heart is not a good guide since it frequently stops without any preceding slowing or irregularity, as was often found in the present series.

At least 4 of the infants lost might have been saved had the membranes been ruptured earlier. The tell-tale presence of meconium would have obviated the necessity of relying entirely on the foetal heart for indications of foetal distress.

When meconium is associated with accidental haemorrhage or pre-eclamptic toxaemia, it is also particularly dangerous. This has been the experience of Walker[23] as well. The presence of meconium early in labour is an ominous sign. In most of the cases in which infants were lost in the present series meconium was present for 6 or more hours before delivery. The advisability of allowing a trial of labour to continue in the presence of meconium, particularly frank meconium, is highly debatable.

Probably the most difficult cases to manage are those in which meconium is first noticed fairly late in labour (after the os is 3 fingers dilated). No one can be blamed for hesitating to terminate labour at this late stage by Caesarean section, particularly if everything had been progressing smoothly. Conservatism may be justifiable, but only in the absence of those complicating factors referred to previously. The necessity for a forceps delivery at full dilatation cannot be denied, particularly in the presence of frank meconium. Shortening the second stage can be of extreme importance in preventing further embarrassment of an already hypoxic infant.

No doubt numerous experienced obstetricians could quote the vaginal deliveries which they effected time and again in spite of the presence of meconium and without any undue foetal loss. The present series, although admittedly small when broken down into its numerous sub-divisions, nevertheless clearly shows the increased foetal loss associated with the presence of meconium. As a pilot survey for a much larger series, it can hardly be ignored because of the facts it brings to light. If the scheme of treatment suggested in this paper had been followed, the outcome might have been like this:

In the 169 cases in which meconium was present, an extra 12 Caesarean sections would have been carried out. In addition, 2 Caesarean sections would have been done earlier, and a further 19 forceps deliveries carried out. This added amount of intervention might have saved 8 of the 15 salvable infants who were lost. Whether this less conservative attitude is justified in order to achieve the higher foetal survival rate, is the decision which each obstetrician must make for himself when faced with the problem.

SUMMARY

1. 169 cases, in which meconium was present in association with vertex presentation, are analysed. These occurred among 2,600 consecutive deliveries. This is a pilot survey for a much larger series.

2. The perinatal mortality associated with the presence of meconium is discussed.

3. The diagnostic value and significance of meconium as an indicator of foetal distress in this series is analysed. The outcome of the various cases is discussed in relation to (*i*) the dilatation of the cervical os at the time when meconium was first diagnosed and (*ii*) the method of delivery.

4. A markedly increased foetal mortality was found where meconium was present for more than 6 hours before delivery.

5. Throughout, *the presence of thick or frank meconium is shown to be associated with a higher foetal mortality than is the case with thin or light-green meconium staining of the liquor amnii.*

6. There was a surprisingly low incidence of congenital abnormalities in the present series (2·4%). A conservative attitude with regard to Caesarean section because of the fear of delivering a congenitally abnormal child is not justified.

7. A scheme for the treatment of vertex deliveries associated with meconium (this being based on a retrospective analysis of the present series) is outlined. Early Caesarean section, in order to prevent possible foetal loss, is recommended, particularly in the presence of frank meconium. If other signs of foetal distress or certain associated complicating factors are present, early intervention is also indicated. At full dilatation, delivery by forceps is necessary.

I should like to thank Prof. J. T. Louw for his encouragement and his help with this investigation; also Dr. Fred Benjamin for his assistance in the preparation of this paper. Acknowledgements are due to Dr. G. J. Joubert, Superintendent of the Somerset Hospital, Cape Town, for permission to publish data about the patients.

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HEARTBURN IN PREGNANCY

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Heartburn is a common complaint in pregnancy, occurring in approximately 60% of women.[1] It is often looked upon as one of the minor discomforts to be endured by the expectant mother without any attempt being made to ascertain a cause for it. This condition should not always be considered lightly; besides being, very often, a source of great discomfort to the patient, it may be a warning of any one of several more serious conditions.

In 85% of cases, the onset of heartburn is between the third and fifth month of pregnancy. The symptoms are most severe between the sixth and eighth months in 45%, and in the ninth month in 20%. During the last three weeks of pregnancy, symptoms cease spontaneously in 75% of patients.[1]

AETIOLOGY

Pregnancy exerts a disturbing influence on certain functions of the alimentary tract. This is shown by the effect of pregnancy on peptic ulcer. The causes of heartburn, be they endocrine, biochemical, reflex or mechanical, remain theoretical in many cases, but a definite cause can be found if sufficient thought and care is given to the establishment of a diagnosis.

Alterations in Gastric Acidity

Formerly, heartburn was blamed on hyperchlorhydria, but alkalis give inconstant relief. In many pregnant women there is a notable reduction in the free and total gastric acidity, particularly during the second trimester. Small doses of dilute hydrochloric acid have, therefore, been used, but again with variable success.

Reflux of Gastric Contents into Oesophagus

Williams[2] reported that, in addition to reduction in acidity, there is a diminution of gastric motility in pregnancy, and it has been suggested that this, together with a neuromuscular disturbance at the cardio-oesophageal junction, allows regurgitation into the oesophagus with resultant oesophagitis. Also, the stomach is elevated and rotated as the uterus ascends in pregnancy, and delayed emptying occurs which is probably the result of atony rather than a mechanical cause. The atony of the stomach is part of the generalized muscular atony that occurs in the hollow organs during pregnancy, from hormonal changes. This condition of the stomach helps in the regurgitation of stomach contents into the oesophagus. This is well illustrated in the following case report:

Mrs. R. M., aged 28 years, para 2. Two full-term normal deliveries. History of marked heartburn with the second pregnancy; no heartburn during the first pregnancy. Attended antenatal clinic from the 14th week of the present pregnancy. Developed severe heartburn from the 18th week, which was not relieved by alkalis or hydrochloric acid, but became progressively worse. A barium meal was carried out at the 30th week of pregnancy to exclude peptic ulcer or hiatus hernia.

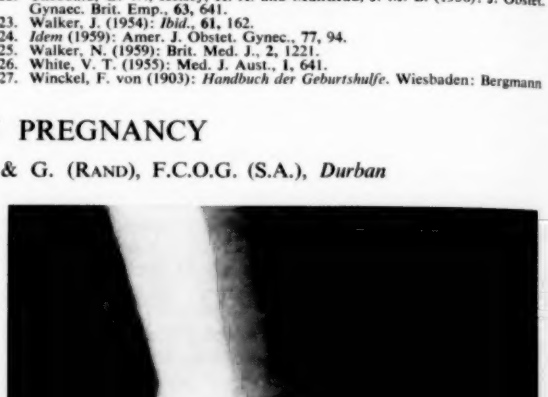

Fig. 1: Barium meal showing free reflux of barium from the stomach into the oesophagus, with the patient supine.

X-ray report (Fig. 1). In the supine position the patient had marked free reflux of barium from the stomach into the oesophagus. There was no evidence of hiatus hernia, peptic ulcer or ulceration at the lower end of the oesophagus.

A repeat barium meal, three months after delivery, showed that in the Trendelenburg position there was no oesophageal regurgitation or hiatus hernia. This readily explains how pyrosis occurs, owing to reflux of gastric contents into the oesophagus, especially in the recumbent position. Altering the gastric contents from acid to alkali in these patients helps little in reducing symptoms. Treatment with 'prostigmin' is rational since the drug has been shown, both by experimental observation and clinical trials, to produce increased motility of the stomach.

Peptic Ulcer

The course of chronic gastric and duodenal ulcer is said to be improved during pregnancy. Peptic ulcers are rare in pregnancy, but the reasons for this are uncertain. There is a fall in the gastric-acid content in pregnancy as mentioned above, and this may play a part. Way[3] showed that an inverse relationship exists between the concentration of chorionic gonadotrophin in the urine and of free acid in the gastric juice. The presence of free bile in only 2 of the 63 cases he investigated is a point against regurgitation of duodenal contents causing a diminution of gastric acidity. Endocrinal factors may play a part in the prevention of peptic-ulcer formation. Its predominance in men, its milder course in

women, and its rarity during pregnancy, suggest that gonadotrophins or oestrogens and progestogens exert an inhibitory influence on ulcer formation. Sandweiss *et al.*[4] have shown that chorionic gonadotrophins will prevent the formation of jejunal ulcers in dogs in whom the duodenal contents had previously been shortcircuited directly into the lower ileum. The rôle played by the increased output of adrenocorticotrophic hormone and the corticosteroids in the course of peptic ulcer in pregnancy is ambiguous, since peptic ulcer is a contraindication to their use in clinical practice. Also, in conditions bringing about a marked stress reaction with an increased output of corticosteroids, such as burns, duodenal ulcer may appear.

The misdiagnosis of the presence of a peptic ulcer or a perforated peptic ulcer during pregnancy may easily lead to a fatal outcome. As with a perforated appendix in pregnancy, the symptoms and signs may be masked. The signs of subsequent peritonitis may be obscure, probably owing to the high corticosteroid levels in pregnancy bringing about a lack of physical response to inflammation. Although peptic ulcer is rare in pregnancy, its presence must always be borne in mind where pyrosis is a presenting symptom.

Hiatus Hernia

Hiatus hernia is four times more common in women than in men. It is difficult to assess its frequency during pregnancy and there is not much reference to its statistical occurrence. A large-scale investigation is required over a long period.

Herniation of part of the stomach through the oesophageal hiatus is commonly overlooked. In pregnancy it is precipitated by a rise in intra-abdominal pressure, by recumbency and by laxity of the tissues of the oesophageal ring, caused by hormonal changes in pregnancy. Congenital predisposition plays an important part and is precipitated by pregnancy.

The symptoms are protean and may consist of epigastric discomfort, vomiting, especially late in pregnancy, haematemesis, retrosternal pain, and dysphagia. Resultant oesophagitis due to reflux is common, but ulceration and stricture formation are rare in hiatus hernia occurring in pregnancy. The symptoms are aggravated by recumbency and bending forward, and are worse in the last trimester and tend to clear up shortly after delivery. The presence of hiatus hernia is an important consideration in heartburn and vomiting late in pregnancy. The patient may go through pregnancy and labour uneventfully and many such cases are not diagnosed.

Large hernias can also be the cause of dyspnoea and a tendency to cyanosis in labour. Murless[5] has pointed out that the patient can be tided over delivery without resorting to radical measures, giving small frequent meals, inducing labour if symptoms become worse, and relieving effort in the second stage of labour by forceps delivery under local analgesia. The adoption of the high sitting position in bed at night affords much relief of symptoms. After delivery symptoms disappear and the hernia cannot be demonstrated in most patients. Sometimes, however, the hernia persists after delivery, especially in the older multipara. In those patients in whom the hiatus hernia cannot be demonstrated after delivery, it is important to realise that recurrence is likely later in life when the patient tends to become fatter.

The following case reports illustrate these points:

1. Mrs. A. J., aged 27 years, para 3. History of heartburn with the last pregnancy. Developed severe heartburn during the present pregnancy at the 20th week. The symptoms were not relieved by alkalis. Barium-meal examination was carried out at 32 weeks and a hiatus hernia was clearly demonstrated; there was no evidence of ulceration or stricture formation. On re-X-ray 2 months after delivery there was no trace of a hiatus hernia.

2. Mrs. J. R., aged 33 years, para 2. History of heartburn with the first 2 pregnancies. Attended antenatal clinic at 20 weeks during the present pregnancy, complaining of severe heartburn and vomiting. No haematemesis. Barium-meal examination showed no ulceration of the oesophagus or stomach. The duodenum, however, showed some oedema, with a small ulcer crater within this. This appearance was constant throughout the examination. In addition, when the patient was placed in the Trendelenburg position a small amount of gastric mucosa was seen to prolapse through the oesophageal hiatus into the thorax. This disappeared while the patient was swallowing barium, but re-appeared again after the swallowing was completed.

Conclusion: A small hiatus hernia could be seen and an active duodenal ulcer was present.

Repeat X-ray one month postpartum: As in previous X-ray, a hiatus hernia was again demonstrated and could be seen prolapsing past the diaphragm. The small duodenal ulcer was no longer visible (the patient had been given treatment for her peptic ulcer before and after delivery).

DISCUSSION

Heartburn should not be accepted as one of the discomforts of unknown aetiology that occur in pregnancy. If further investigations are carried out, a cause will very often be found, and this may lend itself to treatment or give warning of serious pathology that may become worse later in life. The regurgitation of stomach contents into the oesophagus is far more frequent than was previously thought.

That peptic ulcer is rarely associated with pregnancy is true, but its presence must not be dismissed without thorough examination, since a misdiagnosis may lead to a fatal outcome. It should also be borne in mind that peptic ulcer may occur in association with hiatus hernia.

Hiatus hernia is a common serious condition and is precipitated by pregnancy at an earlier age in those patients who are congenitally prone to it. Where hiatus hernia disappears after pregnancy it will probably manifest itself again at a later age when the patient's weight increases. A sliding hiatus hernia is the commonest—hernia *en glissade*. This is an important point because its presence produces incompetence of the cardia and this allows reflux of stomach contents into the lower part of the oesophagus.

SUMMARY

1. Heartburn in pregnancy is discussed with its various causes. It is a common condition occurring in approximately 60% of women.

2. Heartburn in pregnancy should not be considered lightly, and is not a normal discomfort that the expectant mother must tolerate. Very often a cause will be found if sought. It may be the presenting symptom of more serious conditions.

3. In many pregnant women there is a notable reduction in the free and total gastric acidity.

4. Reflux of gastric contents into the oesophagus is common during pregnancy and probably results from a diminution of gastric motility and delayed emptying, together with a neuromuscular disturbance at the cardio-oesophageal junction. These disturbances are partly mechanical in nature, ascent of the uterus causing elevation and rotation of the stomach. Hormonal changes also play an important rôle.

5. Hiatus hernia is four times more common in women than in men, and is not uncommon in pregnancy. It occurs in

those patients congenitally prone to it and is precipitated at an earlier age as a result of pregnancy. It may no longer be demonstrable after delivery, but is likely to manifest itself again at a later age, when a tendency to excessive weight gain occurs.

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'ACTIFED'—A CLINICAL IMPRESSION

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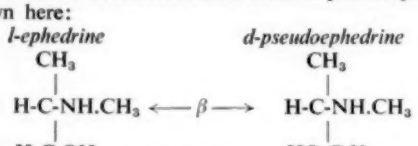
Some years ago I described a series of cases under the title 'Asthma in young boys' in this *Journal*.[1] I then showed that a combination of drugs, such as pseudoephedrine and an antihistaminic, should have most beneficial effects if given to asthma sufferers every night before going to sleep, provided it was done over long periods. Messrs. Burroughs Wellcome and Co. Ltd., taking cognizance of my article, informed me that they, too, had investigated the effect of such a combination of drugs in patients in whom the upper respiratory tract was involved idiopathically, or through allergy or infection.

The commercial name given to this combination of drugs is 'actifed'. It contains 30 mg. of pseudoephedrine, and 1·25 mg. of triprolidine hydrochloride which is a powerful antihistaminic. The pseudoephedrine combination is interesting; I find it is less toxic and has fewer side-effects than ephedrine hydrochloride. Also it has shown less central stimulation and vasopressor effect. I particularly wish to give full details of this drug as found in the literature, because I feel that it has not been sufficiently investigated and could be of great benefit where a substitute for ephedrine hydrochloride is sought, especially in view of the fact that it is less toxic and has less side-effects.

D-PSEUDOEPHEDRINE

Ephedrine contains two asymmetric carbon atoms and therefore has 6 synthetic isomers, viz. *l*, *d* and *dl* ephedrine, and *l*, *d* and *dl* pseudoephedrine.

Two of these also occur naturally—*l*-ephedrine and *d*-pseudoephedrine—and the chemical difference between them lies in the relation between the hydroxyl group on the α- and the methylamino group on the β-carbon atom of the side chain, the groups being close to each other in ephedrine and removed from each other in pseudoephedrine, as shown here:



d-Pseudoephedrine, the natural alkaloid, is used in actifed because it is almost as potent, weight for weight, as ephedrine, but produces neither the same degree of central stimulation nor the vasopressor effect. The 1949 *British Pharmaceutical Codex* states: '*d*-Pseudoephedrine resembles ephedrine qualitatively, but its effects, particularly on the blood pressure, bronchi and higher centres, are weaker. It is less toxic and has fewer side effects'.

Action

d-Pseudoephedrine is an orally effective nasal decongestant which takes effect 15 - 30 minutes after oral administration. The mucous membrane of the entire respiratory tract is gently, but promptly, decongested through action on sympathetic nerves and smooth muscle. With its simultaneous bronchodilating action, *d*-pseudoephedrine facilitates free breathing by opening the airway from nostrils to alveoli. The systemic route of administration obviates the use of topical preparations, thereby protecting the delicate inflamed nasal mucosa from additional trauma. Furthermore, it gives desirable prophylaxis against middle-ear complications by reaching and decongesting the Eustachian tubes.

Nasal Decongestion

d-Pseudoephedrine restores patency of the nasal passages by shrinking the engorged nasal mucosa and quickly eliminating tissue hyperaemia and oedema. 'Its dilator action on the bronchioles, as well as its contracting action on the mucous membrane of the nose, do not essentially differ in potency from that of ephedrine'.[2]

Bronchodilation

In clinical use,[2,3] and in laboratory studies,[4] the bronchodilator action of pseudoephedrine appears to be quite marked. Complementing the bronchodilating action, pseudoephedrine decongests the mucosa of the tracheobronchial tree.[2,3]

Toxicity

In animal studies pseudoephedrine has been found to be nearly 50% less toxic than ephedrine. The LD_{50} for mice is 700 mg. per kg.

CASE REPORTS

Of 30 patients for whom I prescribed actifed, the majority responded favourably and could go about their duties undisturbed. These patients suffered from allergic rhinitis, bronchial catarrh, asthma, sinusitis, postnasal drip, hayfever and Eustachian-tube blockage. Also, there were patients with chronic disease of the upper respiratory tract, where symptoms had persisted despite operation or bombardment with antibiotics. My trial has now continued for a period of just over a year, and I propose to describe in brief some patients for whom I have prescribed actifed.

Case 1

European male, aged 70 years: He had a history of sinus infection for 10 years, and was treated with antibiotics and sinus puncture with incomplete relief. His condition gradually developed into a persistent bronchitis and bronchial catarrh as well as ?septic arthritis. Sensitivity tests were done against organisms from his sputum, and so many antibiotics were used in an attempt to clear his upper respiratory tract that he developed moniliasis. The sinusitis and bronchitis persisted and eventually cortisone was tried as well. He was given actifed, 1 tablet twice daily, and thereafter was found to be completely relieved of bronchial catarrh and wheezing chest as well as postnasal drip. He still expectorates a small amount of sputum in the morning. At present he finds

that 1 tablet on alternate mornings is enough to control his symptoms.

Case 2

European male, aged 49 years: He came from the Congo to live in Sea Point, but experienced severe chest wheezing at night, a painful dry cough and nasal blockage. During the day he was quite normal. He had never had any illness like this before, except bronchitis 2 years previously. On examination he had typical bronchospasm. At first it was necessary to give him an injection of adrenaline, but I decided to give him actifed tablets, 1 twice daily for 2 days and thereafter 3 times a day. With the exception of slight drowsiness his symptoms were completely relieved.

Case 3

European male, aged 40 years: He complained of deafness of his left ear, which had wax in it, but despite syringing there was no improvement. In addition he had an old-standing suppurative otitis media of his right ear. I referred him to an ear, nose and throat specialist who diagnosed a blocked left Eustachian tube. He politserized him without improvement. I decided to try actifed, and within 5 days his Eustachian-tube deafness had completely cleared up. In the patient's own words 'it simply was wonderful!'

Case 4

European male, aged 36 years: He consulted me in 1956 with a diagnosis of catarrh of the upper respiratory tract. Sensitivity tests showed him to be sensitive to house dust and the pollen of flowers. At this time he was using an adrenaline spray as well as antihistaminics, but this treatment made him very tired and depressed. He had moved from where he lived previously to Sea Point. In addition he had developed a bronchospasm at night. Further treatment with antibiotics, an electric air filter and warmer in his room, plus sprays and antihistamine did not relieve him. I suggested that he should try actifed, and for the past 6 months he has continued to use it. It has helped him more than any other treatment he has had up to date. He stated that he could now sleep undisturbed at night and that his bronchospasm and nasal blockage are definitely relieved.

Case 5

European girl, aged 8 years: She had had nasal disturbances since she was 5 years old. She had a tonsillectomy, but this did not improve matters. Sensitivity tests with antibiotics, inhalations and breathing exercises were also tried, but they brought little relief to the nasal disturbances. An X-ray revealed definite maxillary-sinus involvement. It was necessary to have a sinus operation performed, despite her age. There was some improvement, although the child was troubled at school because her teacher continually reprimanded her for blowing her nose. I prescribed actifed for her and found that her nasal disturbances were gradually controlled by its use. Also, it helped to tide her over until she could have a further operation to her sinuses, which have since cleared up.

Case 6

European female, aged 20 years: She suffered from bronchospasm at night. Her symptoms were nose blockage, slight cough, and a general tightness of her chest. Despite the use of 'amesec' capsules, adrenaline injections and desensitization with azoprotein, I was obliged eventually to prescribe cortisone to relieve her symptoms for 6 weeks. When the course of cortisone was completed, her symptoms gradually came back. I found that in prescribing actifed, in addition to a much smaller dose of cortisone than formerly, I managed to control her symptoms for the last 6 months.

Case 7

European male, aged 70 years (seen by Dr. J. Sonnenberg): He states that for the past 6 months his nose just ran without warning. He also developed bronchospasm. His nose was cauterized, but this gave only temporary relief. On examination he appeared to have typical allergic rhinitis. He was given actifed, 1 tablet twice daily, with almost dramatic improvement.

CONCLUSION

The 7 patients I have mentioned represent a fair cross-section of my trial cases helped by actifed treatment. It is not a cure, except in seasonal cases, but it is certainly a very good preparation in the symptomatic treatment of upper respiratory conditions, especially when a patient has been through a long period of illness with many different forms of treatment and the doctor is rather at a loss for something new. I have noticed no side-effects, except slight drowsiness at the beginning of treatment. This passes off after a few days' treatment.

I must point out that actifed is not a blunderbuss combination, containing only pseudoephedrine and triprolidine. If an antipyretic is necessary, aspirin compound could be given in addition.

Since this article was prepared, Burroughs Wellcome have brought out actifed tablets in half the strength used in this trial. This will do away with the drowsiness and allow for greater flexibility in dosage.

I wish to thank Prof. F. Forman, Prof. N. Sapeika, and Dr. I. Grayce for kindly reading this paper and for their useful comments. I also wish to thank Burroughs Wellcome and Co. (S.A.) Ltd. for supplying the tablets used.

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SELF-RADIATION PRODUCTS FORMED FROM 3,5,3'-TRI-IODO-L-THYRONINE LABELLED WITH ^{131}I*

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3,5,3'-tri-iodo-L-thyronine, labelled with ^{131}I is used in our laboratories mainly for two purposes, namely, for metabolic studies and for red-cell uptake as an additional indicator of thyroid activity.[2] Because of its marked influence on metabolism, low concentrations of the thyroid hormone are required for metabolic studies. This necessitates the preparation of thyroid hormones of extremely high specific radioactivity. The test for red-cell uptake depends upon the affinity of red cells for thyroid hormones. This affinity is less than that of the serum proteins. As thyroid activity increases, increased concentration of circulating thyroid hormone causes saturation of the binding protein in serum, and as a result more and more hormone is taken up by the red cells. Since red-cell uptake therefore clearly depends on the concentration of the hormone, a small concentration of the hormone with high radioactivity is again required.

In South Africa we still depend largely on the import of labelled hormones of high specific radioactivity. These hormones reach our laboratories, at the earliest, 5 days after production (day of production is taken as the day of determination of specific activity) and sometimes only a few days before the expiry date (14 days after production). During transport and storage, radiochemical decomposition from self-radiation[3] may occur. It is conceivable that the extent of decomposition bears a relationship to the specific radioactivity.

* Abstract of a paper presented at Research Forum, University of Cape Town.

The possible processes leading to the formation of artefacts are as follows:

(*a*) De-iodination which could result in the formation of ^{131}I, 3,5-di-iodothyronine and, less probably, to 3-mono-iodothyronine.

(*b*) The oxidation of ^{131}I to $^{131}I_2$ which could re-iodinate the de-iodinated thyronines to labelled tri-iodothyronine and thyroxine.

(*c*) The introduction of chemical changes in the alanine side-chain which would result in the formation of the lactic acid analogues.[3]

In this study, 3,5,3′-tri-iodo-L-thyronine, labelled with ^{131}I, was analyzed quantitatively for the formation of decomposition products at fortnightly intervals up to 75 days by chromatographic, electrophoretic, and radio-autographic means. The hormone was obtained from Abbott Laboratories, Oak Ridge, Tennessee, USA, labelled by a method of radio-isotopic exchange[1] with ^{131}I in the 3′-position only. The specific radioactivity of the samples used varied from 21·4 to 29·6 mc/mg., and the concentrations from 13·0 to 19·5 μg. of tri-iodothyronine/ml. in 50% propylene glycol.

The 4 major radiochemical breakdown products formed were ^{131}I, ^{131}I-labelled 3,5-di-iodothyronine, ^{131}I-labelled thyroxine, and an hydroxy-acid analogue of tri-iodothyronine or of di-iodothyronine, or both. It was established that the chromatographic solvents used in this study were not responsible for these breakdown products. Quantitative analyses indicate that the mechanism of radiochemical decomposition of ^{131}I-labelled tri-iodothyronine proceeds as follows:

(*i*) De-iodination of the most labile position (3′-position).

(*ii*) ^{131}I exchange at positions of 3 and/or 5 which renders the 3,5-di-iodothyronine radioactive.

(*iii*) Oxidation of ^{131}I to $^{131}I_2$.

(*iv*) Iodination of ^{131}I-labelled tri-iodothyronine to labelled thyroxine.

(*v*) Oxidative de-amination followed by reduction of the side chain.

It is concluded that the danger exists of interpreting a self-induced radiation product as a metabolic transformation. Furthermore, if a stored sample of ^{131}I-labelled tri-iodothyronine is used for studies of red-cell uptake, a correction factor is required.

In our laboratory we now prepare our own ^{131}I-labelled thyroid hormones. The main difficulty is encountered with the specific radioactivity determination in samples in which the chromatographically pure product is too small to be weighed accurately. To overcome this problem we determine the concentration of purified thyroid hormone by stable iodine (I^{127}) analyses, and the radioactivity (^{131}I) by counting.

REFERENCES

1. Gleason, G. I. (1955): J. Biol. Chem., **213**, 837.
2. Hamolsky, M. W., Stein, M. and Freedberg, A. S. (1957): J. Clin. Endocrinol., **17**, 33.
3. Tata, J. (1959): Clin. Chim. Acta, **4**, 427.

THE ECONOMICS OF MEDICAL PRACTICE*

H. M. SEGALL, *President, Border Branch (M.A.S.A.) 1960*

The practice of medicine deals with the maintainance and improvement of the health of the human being and the preservation and prolongation of human life. With this as his stock in trade, it is obvious that the individual who follows the practice of medicine as a career, is swayed primarily by a sincere mission to serve his fellowmen, and is therefore essentially an altruist and an idealist at heart. It is most unfortunate that the doctor must earn a living in the pursuit of this ideal, for material gain is not the driving force that attracts an individual to medicine, but it is unfortunately something that strongly affects his ability to pursue that ideal. For the doctor today, as a result of the rising cost of living, has been forced to consider the commercial aspect of medical practice. As a business proposition the practice of medicine is the most difficult and demanding method of earning a living; and, if the economic side alone is considered, the capital involved is not justified by the return. There is no more onerous way of earning a living — for the individual doctor cannot devote the time he should to his family, to outside pursuits, or to the necessary holidays and relaxations to maintain his health. It is a precarious existence for the first ten years of practice, when so much can go wrong at a time when, economically, it is impossible to provide adequately for unforeseen circumstances.

Dr. Segall

It is therefore necessary, in this material age, for the doctor to discuss and control payment for his services and to deal with the business side of medicine, so that he can follow the practice of medicine and provide adequately for the maintenance and upbringing of his family, the necessities of daily living, and a means of retirement.

With the awakening of the general public to the realization that their most valuable possession is good health and not ownership of a business, a house, or a car, has come the desire to ensure the preservation of this asset. The man in the street now wants to insure not only his business, his house, and his car against fire, theft, and accident, but also himself against ill-health.

Earlier on, the profession had lent its aid in providing a means of payment for medical services for those who desired to have their own doctor, but who could not afford private fees, in the form of being a party to medical benefit societies and later medical aid societies. This inevitably opened up the way to heavier demands on the profession in the way of subsidization — not only to provide, but also to pay for the health of the individual. No other profession or calling agrees to the charging of a different fee for the same commodity — fees in general vary according to what the buyer can afford to pay.

With the establishment and recognition of the medical insurance schemes, a dangerous principle has been adopted — that of subsidizing private concerns. This is the present unrealistic state of affairs and it is felt by a large number of members that it is not fair to ask any profession to subsidize private enterprise, if that profession in turn cannot obtain any subsidy for itself. Having fixed a reasonable and just fee for its services, the profession should cease to recognize any schemes at all that demand discounts or subsidies. If this stand is taken, then a member of the medical profession can follow his calling in a way that, like any other calling, will allow adequate provision for the needs of himself and his family, and not make it necessary to continue to work at all hours until the age of seventy or even longer.

Many members of the Association feel that our approach is impractical and that it creates a dangerous precedent. Unless a principle is adopted that does not ask the profession to attend to the health of the public and in addition help pay for that service, the idea of fixing a ceiling for members of the public who are insured will grow out of bounds, and the Association will no longer speak for a majority of practitioners in this country. Economically the harassed doctor will be driven to take independent action, and this will expose the profession to a very real threat of state medicine — a threat to which an organization such as ours (a voluntary body) has no answer.

If, on the other hand, it is felt by the majority of members

* Extract from Valedictory Address, East London, 11 February 1961.

that the payment of doctors *is* the concern of the Association, then the answer is to establish, on a non-profit basis, an insurance scheme which will cover all groups of the public, but which will remain under the control of the Association. The nucleus of such a scheme exists in the Transvaal in the form of the Medical Services Plan which is sponsored by the Association. The extension of this Plan, or even the handing over of the scheme to an insurance company to run under the control of a board on which the majority vote will be in the hands of doctors, would not only adequately provide an efficient service, but would guarantee that there could be no interference by any private business concern, and that the economic side of medical practice would not deteriorate any further. To achieve this would mean some considerable reorganization of our Association — a step that should result in giving the profession a better status than it enjoys today.

I feel that the time has now come for our organization to appoint a trained business manager whose duties will be to deal with the economic side of medical practice. I hope that the near future will see this ideal materialize.

VYFDE AKADEMIESE JAARDAG VAN DIE KARL BREMER-HOSPITAAL EN DIE FAKULTEIT VAN GENEESKUNDE VAN DIE UNIVERSITEIT VAN STELLENBOSCH

Die jaarlikse akademiese jaardag sal op Woensdag en Donderdag 9 en 10 Augustus 1961 gehou word in die Burgersentrum, Bellville, Kp. Die volgende is die program:

REFERATE

Woensdag 9 Augustus

2.00 nm. Offisiële opening: Prof. F. D. du T. van Zijl.
2.10 nm. Die Operasiesaaltegnikus: Dr. R. L. M. Kotzé.
2.25 nm. Die Invloed van aluminium en kalsiumfluoried op die longe en die respiratoriese ensieme van proefdiere: Dr. F. M. Engelbrecht.
2.45 nm. Diabetiese osteo-artropatie: Dr. P. D. R. van Heerden en dr. C. L. Wicht.
3.00 nm. Die moontlike verband tussen die α selle in die pankreas-eilandweefsel en die cholesterol-metabolisme: Dr. A. C. Esterhuizen.
3.15 nm. Teepouse.
Filmvertonings gedurende teepouse:
1. The iron cycle as revealed by the electron microscope.
2. Chronic cor pulmonale in the emphysematous patient.
4.00 nm. Die dik been: veneuse inkompetensie: Dr. J. J. W. van Zyl.
4.30 nm. Oöforektomie en die menopouse: Dr. R. W. A. Nel.

Donderdag 10 Augustus

9.00 vm. Plaaslike laboratorium-ondervindings met *Staphylococcus aureus:* Dr. H. H. D. Brede.
9.30 vm. Urologiese probleme by kinders: Dr. J. D. de B. Joubert.
10.00 vm. Tetanus neonatorum: 'n voorlopige mededeling aangaande behandeling: Dr. M. P. Keet en dr. A. B. Murray.
10.15 vm. 'n Kaapse velsiekte: papulêre urticaria: Dr. James Marshall.
10.30 vm. Teepouse.
Filmvertonings gedurende teepouse:
1. Surgical treatment of otosclerosis.
2. X-ray cinematography: A modern technique of diagnostic radiology.
11.00 vm. *Groepbespreking:* ‚Hoë bloeddruk'.
Moderator: Dr. C. L. Wicht.
Internis: Dr. P. W. A. Botha.
Uroloog: Dr. J. N. de Klerk.
Ginekoloog: Prof. J. N. de Villiers.
Patoloog: Prof. H. W. Weber.
Nierbiopsie-studies:
12.00 nm. Tegniek en kliniese aspekte: Dr. P. W. A. Botha.
12.15 nm. Patologiese aspekte: Dr. S. S. Grové.
12.30 nm. Middagete.
Filmvertonings gedurende die etensuur:
1. Adrenaline and noradrenaline.
2. The story of oxytocin.
2.00 nm. Besigheidsvergadering en verkiesing van Nuwe Komitee.
2.30 nm. Voorkoming van postpartum bloeding: 'n 4-jaar analise: Prof. J. N. de Villiers.
2.45 nm. Die insidensie van ekstrakraniale vatafsluiting as oorsaak van hemiplegie: Dr. M. A. de Kock.
3.00 nm. Karsinoom van die prostaat: Dr. A. J. S. Burger.
3.30 nm. Teepouse.
Cine-endoscopy.
4.00 nm. Abdomino-mediastinotomie: Dr. B. J. van R. Dreyer.
4.15 nm. Die urologiese aspekte van hiperurisemie: Dr. J. N. de Klerk.
4.30 nm. Vaginale histerektomie: Dr. P. F. M. du Toit.
4.45 nm. Perikarditis: Dr. D. G. le Roux.

WETENSKAPLIKE UITSTALLINGS

Biopsie-tegnieke in gebruik in die departement van interne geneeskunde	Departement van Interne Geneeskunde.
Elektron-mikrograwe van pankreas-eilandweefsel	Dr. A. C. Esterhuizen, Departement Fisiologie.
Die gebruik van kunshars in die montering van delikate museummonsters	Mej. H. I. le Roux en mnr. N. H. F. Harrington, Departement Anatomie.
Eksfoliatiewe sitologie: Interessante gevalle waar sitologie van diagnostiese waarde was	Sitologie-afdeling, Departement van Ginekologie en Verloskunde.
Aorto-plasentografie	Dr. J. M. M. Basson en dr. G. D. Burger.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. C. M. Ross, of Pretoria, is leaving on a short visit to dermatological clinics in the UK and Israel. He will return on approximately 10 August 1961.

* * *

Mr. Gerald Kane, F.R.C.S., has returned to Durban after an overseas visit of 12 weeks during which time he visited gastro-enterologic clinics in Britain, Holland and Sweden.

Mr. J-J. Brossy, has returned to Johannesburg after reading a paper on 'Major colon surgery in geriatric patients' at the American Proctological Society Meeting in Pittsburg. He also visited medical centres in the United States and Great Britain.

* * *

Dr. A. Robins, dermatologist, of Johannesburg, has resumed his practice after a recent visit overseas.

South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting. The next meeting will be held on Monday 7 August at 5.10 p.m. in the Institute Lecture Theatre. Mr. H. Patterson will speak on 'Entomological aspects of arbor virus research'.

* * *

Research Forum, University of Cape Town. The next meeting will be held on Thursday 3 August at 4 p.m. in the Tutorial Room of the Pathology Department, Medical School, Observatory, Cape. Dr. W. D. Marais will speak on 'Some aspects of placental pathology with special emphasis on its relationship to the toxaemias of pregnancy and spiral atherosclerosis'. This will be followed by a Staff Clinical Conference at 5.10 p.m. in the Falconer (E-floor) Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. All who are interested are invited to attend these meetings.

Clinico-pathological conferences will be held, in conjunction with the Department of Pathology, on Thursdays (other than those scheduled for Research Forum) at the same time and venue and are followed by staff clinical conferences at 5.10 p.m. All doctors who would be interested in attending staff ward rounds should communicate with the Secretary of the Department of Medicine.

* * *

Eastern Transvaal Branch, (M.A.S.A.) Spitz Golf Trophy. A very successful day was enjoyed by members of the Eastern Transvaal Branch of the Medical Association at Standerton on Sunday 18 June 1961.

The prize-winners were as follows: *Spitz Cup:* 1. Mr. Van der Berg, Mr. Dekker. 2. Dr. L. de Villiers, Dr. U. van Heerden. 3. Mr. Edwards, Mr. Beeton. 4. Dr. Joffe, Mr. Saad. 5. Mr. De Waal, Mr. Joubert. *1st Prize Rand Players:* Dr. Maynard, Dr. Kruger. *10th Pair:* Dr. Gough, Dr. Krynauw. *Booby Prize:* Dr. Van Wyk, Dr. Steyn. *2-Club:* Dr. De Villiers, Mr. Beeton, Dr. Bester, Dr. Borgwardt. *Special Prize:* Dr. Bester, Mr. Sadler. *Individual Stableford:* Dr. Spitz, Dr. Trichardt.

The Branch extends its thanks to the following firms for donating the prizes presented: Parke, Davis Laboratories (Pty.) Ltd., B. Owen Jones (Pharm.) Ltd., Bristol Laboratories, Merck, Sharp & Dohme, Lederle Laboratories, Johannesburg Ethical Manufacturers, J. & M. Medical Supplies, S.A. Druggists, Evander Pharmacy, K.P.P., Standerton.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

PHOLTEX

Riker Laboratories Africa (Pty.) Ltd., of Cape Town, announce the introduction of Pholtex, a unique product having two features quite new to pharmacy, and supply the following information:

Pholtex is a long-acting antitussive linctus, giving good control over cough for the whole day (or night) from a single dose. The indications are any type of cough, but particularly the useless, non-productive cough.

In each 4 ml. (one teaspoonful) of Pholtex are 15 mg. of pholcodine as a resinate, and 10 mg. of phenyltoloxamine, also in the resinated form, for prolonged action.

Hitherto long-acting products, with slow or delayed release, have only been available in tablet or capsule form. In Pholtex the resinated active principles are suspended in a special thixotropic vehicle, having a honey-like appearance and generally acceptable flavour. Thixotropy is characterized by the property of a viscous liquid to settle into a gel-like consistency upon standing for a short period. One or two shakes restores normal mobility to the liquid and enables it to be poured readily. The thixotropic property is essential for any suspension containing potent medicaments, since it forms a safeguard against incomplete agitation, which could, in a conventional vehicle, lead to irregularity of dosage.

The pharmacology of pholcodine is too well known to require description, but it may be mentioned in passing that it is free from the undesirable qualities of codein, while having all its virtues. The phenyltoloxamine is a relatively new antihistaminic, possibly one of the most potent known, and yet quite free from any soporific side-effect. The phenyltoloxamine has additional properties which make it of value in an antitussive, namely an antispasmodic action, and a mild local anaesthetic effect which serves to remove irritation in the inflamed larynx. As with most antihistamine substances it also potentiates the effect of the pholcodine.

Although Pholtex so markedly controls useless, unproductive cough, it leaves the function of voluntary cough unimpaired so that there is no interference with the desirable elimination of excess or morbid excretions.

Pholtex may be prescribed for children as well as adults, and is also safe for use by diabetics. Dosage is one teaspoonful, night and morning, for adults, and half to one teaspoonful for children, according to age. Pholtex should never be prescribed in combination, as part of a mixture, since this will destroy the thixotropic property. The pharmacist is also cautioned that it should never be diluted.

Packs are 40 ml. and 120 ml., and the normal prescription should be for the 40 ml. size, which represents nearly a week's treatment.

Literature and trial material are available on request from Riker Laboratories, P.O. Box 3388, Cape Town.

CORTENSOR

Westdene Products (Pty.) Ltd. announce the introduction of Cortensor, the new cardiac preparation from the research laboratories of Dr. A. Wander of Switzerland, and supply the following information:

Cortensor, the simple alephatic amine-6-amino-2 methyl heptanol-(2) (called briefly heptaminol) is primarily a sympathomimetic agent, whose digitalis-like cardiotonic effects considerably outweigh its peripheral vasoconstrictive and central analeptic action. Its most important well-balanced effects are: Intensification of the systolic contraction comparable to that produced by strophanthin G as regards promptness of onset, intensity, and duration; improvement of cardiac blood flow similar to that of current theophylline preparations; resuscitation of the heart muscle, without risk of promoting extrasystoles or cardiac fibrillation; control of arterial hypertension, while blood pressure and pulse-rate remain within normal limits; and intensification of diuresis and only slight stimulation of nervous centres.

These advantages place Cortensor between the highly active cardiac glycosides and other adrenergics with more powerful vasoconstrictive and central analeptic effects.

The range of dose from the threshold of activity to the production of noticeable side-effects (tachycardia, palpitations, etc.) is so wide that dosage can be prescribed without any risks for both prematures and infants and for senile patients.

The therapeutic response to Cortensor lasts for only a few hours and has the advantage of not being cumulative and therefore permitting extremely accurate dosage.

It must be stressed that in severe cases of cardiovascular insufficiency, Cortensor does not replace the more powerful drugs. Nevertheless, Cortensor provides a means of sparing these drugs for emergency cases, of increasing its effectiveness when combined with these drugs, of frequently reducing their dosage and thus improving their tolerability, or of replacing them when they cause pronounced side-effects.

Cortensor is supplied in bottles of 12 and 20 tablets, in drops, 15 c.c., and in injection form, 5 c.c.

Further information may be obtained from the sole South African distributors, Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

WORLD LIST OF FUTURE INTERNATIONAL MEETINGS

Following is a list of international medical meetings to be held during the period January - June 1962. Alterations and additions to this list will continue to be published in the *Journal* from time to time, together with information concerning meetings taking place before January 1962. For the full list of meetings taking place between July - December 1961 see the issue of the *Journal* for 18 February 1961 (35, 136).

Pan American Medical Association, 37th Congress, USA, January 1962. Dr. J. Eller, Director General, 745 Fifth Avenue, New York 22, NY.

Optical Society of America, Meeting, Washington, DC, 15 - 17 March 1962. Dr. Mary E. Warga, Executive Secretary, 1155, 16th St., N.W., Washington 6, DC.

International Association for Bronchology, 12th Congress, Belgium, April 1962. Dr. J. M. Lemoine, Secretary-General of the Association, 189 boul. St. Germain, Paris 7e, France.

Fourth World Congress of Gastroenterology, Munich, 13 - 19 May 1962. Dr. G. A. Martini, Secretary, c/o Universitäts Krankenhaus, Eppendorf, Hamburg, Germany.

International Association for the Study of the Liver, 2nd Meeting, Munich, 19 - 20 May 1962. Dr. G. A. Martini, c/o Universitäts Krankenhaus, Eppendorf, Hamburg, Germany.

Third International Congress of Infectious Pathology, Hamburg, May 1962. Prof. H. Pette, President, Martinstr. 52, Hamburg 20, Germany.

Second Pan American Diabetic Congress, Chicago, May 1962. Dr. B. R. Hearst, 55 E. Washington Street, Suite 1646, Chicago 2, Ill.

BOOK REVIEWS : BOEKBESPREKINGS

METHODS OF STAINING

Staining Procedures, used by the Biological Staining Commission. 2nd edition, revised by H. J. Conn, Mary A. Darrow and Victor M. Emmel. Pp. xii + 289. R4.00. 22½c postage. London: Baillière, Tindall and Cox. 1960.

This book is mainly a selection of staining methods as used in American laboratories. It is not a complete treatise in regard to staining, but it is an excellent help in the standardization of existing methods.

Although the book is not meant to be complete, we would like to see included some classical procedures, such as Neisser's diphtheria staining. Not too much value is given to historical facts, for instance the fluorescence method ascribed to Richards and Miller (1941) was already established by Hagemann in 1937. Repetitions of one and the same method, e.g. description of the fluorescence method on page 229 and again on page 246 could easily be omitted. H.D.B.

GRAFTING WITH POSTMORTEM SKIN

Postmortem Homografts. By James Barrett Brown, M.D., F.A.C.S. and Minot P. Fryer, M.D., F.A.C.S. Pp. vii + 64. 22 figures. R4.40. Springfield, Illinois: Charles C. Thomas. Oxford: Blackwell Scientific Publications. 1960.

In this rather small book the authors make a plea for the use of postmortem skin in the treatment of extensive burns.

It is a very true adage that skin is the best dressing for burns. Parental skin, particularly on the maternal side, is the tissue of election. This, however, involves considerable suffering for the donor and the areas from which the skin has been taken will always be slightly noticeable.

Postmortem skin, though less satisfactory, tends to survive for about 3 - 5 weeks, by which time the patient's general condition has usually stabilized, and further grafting can be carried out with his own skin.

The authors deal with varying methods of preserving the 'bank skin', of which low-temperature freezing appears to be the most satisfactory. N.P.

YEAR BOOK OF OBSTETRICS AND GYNAECOLOGY

The Year Book of Obstetrics and Gynaecology 1960-1961 (*Year Book Series*). Edited by J. P. Greenhill, B.S., M.S., F.A.C.S., F.I.C.S. (Honorary). Pp. 576. Illustrated. $8.00. Chicago: The Year Book Publishers. 1960.

The latest Year Book follows the pattern and high standard of similar previous editions. Summaries of articles published during the past year are grouped under the following headings:

Obstetrics: The evolution of obstetrics, physiology of pregnancy, abortion, ectopic pregnancy, complications of pregnancy, toxaemias, labour, analgesia and anaesthesia, complications of labour, operative obstetrics, haemorrhage of labour, puerperium, and the newborn.

Gynaecology: Diagnosis in gynaecology, infertility, operative gynaecology, infections, benign tumours, malignant tumours, menstrual disorders, and endocrinology.

It is noticeable that more attention is now paid to articles from the European continent, South Africa, and South America. *The Year Book of Obstetrics and Gynaecology* provides a practical way of keeping abreast with the increasing amount of literature. J.N.dV.

WRITINGS OF HENRY SIGERIST

Henry E. Sigerist on the History of Medicine. Edited by Félix Marti-Ibánez, M.D. Pp. xviii + 313. $6.75. New York: MD Publications, Inc. 1960.

Henry E. Sigerist on the Sociology of Medicine. Edited by Milton I. Roemer, M.D. Pp. x + 397. $6.75. New York: MD Publications, Inc. 1960.

Much has been written of the greatness of Sigerist as a medical historian, a medical sociologist and as a man, and it would be superfluous to do more than mention these qualities here. The editors of these two parallel works have chosen a comprehensive selection of his papers and addresses for these anthologies, which reflect not only his subjects but the man himself. They make excellent and stimulating reading. A.H.T.

CORRESPONDENCE : BRIEWERUBRIEK

THE NUMBER OF DOCTORS IN SOUTH AFRICA

To the Editor: This subject has through the years and especially of late exercised the minds of many members of our profession. Opinions seem to differ on whether there are too many, too few, or the right number of doctors in the Republic. In the *Journal* of 12 May 1951, Dr. C. M. Grundlingh stated that there were too many doctors and that the position would deteriorate still further. The figures he quoted as disquieting at that time have become even more disquieting now, ten years later. In the *Journal* of 10 November 1956, *Observer* raised the same question and argued that overcrowding in the profession must soon mean either economic ruin or a state medical service. In the *Journal* of 23 March 1957, Prof. S. F. Oosthuizen, President of the South African Medical and Dental Council, gave a very fair analysis of the same problem, and I think it is worth while quoting from his summary. In his conclusion he said: 'It would appear that in another few years time . . . the position in regard to the total number of medical practitioners in the Union, in relation to the number of persons who require medical services, may become serious.' In another paragraph we read: 'The country is adequately served, and some authorities maintain even more than

adequately served. The emerging problem is one which merits the most serious consideration'. In the same issue of the *Journal*, Dr. Ben Epstein touched on the same problem, but did not reach any conclusions about whether or not the profession is overcrowded.

Lately this issue has again become front-page news, as the result of the survey of the training and employment of medical practitioners in South Africa, prepared by the National Bureau of Educational and Social Research of the Department of Education, Arts and Science. It is a statistical survey and summarizes the present position, also attempting to forecast future trends. According to this survey there is a grave shortage of medical practitioners in the Republic of South Africa, and the position is likely to become worse in the near future. Apparently we will be 2,000 doctors short in 1965.

I think anyone who has studied this survey carefully will find much in it with which he cannot agree, but that is not the point of this letter. Editorial articles in the *Journal* and in *Medical Proceedings* discussed this matter fully, and the Editorial writers are inclined to agree with the findings of the survey. In the final analysis, then, it comes down to this: certain members of the profession (myself included) believe that there are too many doctors in the Republic, and that this overcrowding will become more severe in future. Certain bodies are in agreement with this view. Other persons believe that we are short of doctors, and certain bodies agree with that view. In all this controversy, has anyone ever heard a single pronouncement from the Medical Association of South Africa?* If there has been such an expression of opinion, I cannot recall it. There is endless wrangling about fees for medical services and there are frequent meetings in connection with contract practice, yet how can one ever determine a fair fee for any service unless one considers *supply* (i.e., the number of doctors) as well as *demand?* With regard to the number of doctors in the country, the establishment of new medical schools, the average working life of a doctor, the total number of doctors in relation to the needs of the country — on all these matters the Association has been notably but inexplicably silent. Can it be that this matter does not interest the officials of our Association? Surely this matter is vitally important in relation to the economics of medical practice, about which we hear so much these days.

As a medical missionary, I am not in competition with my fellow practitioners. This is therefore not a case of sour grapes; but I have spoken to many other members of the Association who feel as I do, and very strongly, that a considered and authoritative statement on the number of doctors in South Africa, their economic opportunities, and the future implications of the present rush into medicine, is long overdue from our Association.

Practitioner

9 July 1961

[*The Medical Association is at present engaged in an investigation into this problem; the results will be published in due course — Editor.]

LOCUMS

To the Editor: The issue of the *Journal* for 10 June has just reached me in London, and coincided with a number of replies to my offer in a medical publication of my services as a locum. Today alone there were forty (40) requests!

I have done locums in England, Scotland and Cape Town within the last year and feel obliged to let *Takhaar*[1] know medicine from a locum's point of view.

Firstly, if one is doing only locums for a living, then one's salary, if averaged over the working periods and the enforced non-working periods, is less than R400.00 a month. I say enforced non-working periods since I have never been lucky enough to find one locum ending just where the next begins. Secondly, if *Takhaar* gives examples of carpenters, fitters and turners only, and forgets pianists, or typists, he will continue to be biased in outlook. Thirdly, as exemplified in most hospitals in the USA (where doctors qualify at a higher age and where a greater proportion of them are therefore married), and in locums for general practitioners in Britain, married quarters are taken for granted. Maybe *Takhaar* is the only one in step!

I can only suggest that if locums for general practitioners are doing so well, that he changes from his practice and joins the ranks of those doing locums.

Non-'U'

London
8 July 1961

1. Correspondence (1961): S. Afr. Med. J., **35**, 488.

DIE BEHOEFTE AAN PROFESSIONELE PERSONE

Aan die Redakteur: Met verwysing na u redaksionele artikel[1] waarin u die uitbreiding van mediese opleidingsdienste bepleit, wil ons graag u aandag vestig op die verskaffing van leerkragte.

Benewens die probleem van die verkryging van persone om nuwe poste te beklee, is daar, soos veral in die afgelope paar jaar opmerksaam is, die probleem om diegene wat alreeds hier is, te behou.

Mediese leerkragte wat elders heen gaan, moet seker voel dat daar vir hulle 'n beter toekoms daar wag. Het die tyd nie dalk aangebreek dat die Mediese Vereniging en die Federale Raad die motivering van die persone moet ondersoek en voorstelle formuleer waar dit ook al mag nodig wees nie?

G. J. Rossouw
H. de V. Heese

Groote Schuur-hospitaal
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1. Van die Redaksie (1961): S. Afr. T. Geneesk., **35**, 557.

DISKRIMINASIE TUSSEN PLATTELANDSE EN STEDELIKE PRAKTISYNS

Aan die Redakteur: Die redaksionele artikel in die *Tydskrif* van 8 Julie[1] bring my daartoe om te skrywe oor 'n aangeleentheid wat vir my nie regverdig lyk nie. Ek bedoel u verwysing na die wanverdeling van geneeshere, met 'n plattelandse tekort en 'n stedelike oorbevolking.

Met die wet van vraag en aanbod in gedagte dink ek dan aan die verbasingwekkende beleid van die Federale Raad om, tot die nadeel van plattelandse geneeshere, te diskrimineer tussen die gelde van geneeshere met gelyke opleiding omdat die een op die platteland en die ander in die stad praktiseer. Ja ek weet, daar is die hoër besoekkoste van die stadsgeneesheer. Maar, as die stadsgeneeshere nie in hul onderdele na die platteland stroom om die laer besoekkoste te kom benut nie, dan moet daar tog redes voor wees, wat opvallend nie deur die Federale Raad in ag geneem is nie.

Die redes is onder meer:

Die ontbering van geriewe deur die plattelandse geneesheer, waaronder dikwels elektriese krag; betroubare melk- en watervoorsiening; die vanselfsprekende dienste soos van die tandarts, die wassery, die loodgieter; die skouburg; en verder alle ncemenswaardige sosiale aangeleenthede. Daar is die groot aantal pasiënte op die platteland wat kosteloos behandel moet word, insluitende die verskaffing van medisyne wat die geneesheer self moet koop. Daar is die noodwendigheid van dikwels afgelegde besoeke aan die stad vir private doeleindes oor afstande van honderd myl en meer; en die groter vernieling van jou motor op die plattelandse paaie.

Dit lyk my of die rede vir die diskriminasie eerder geleë is in die feit dat die plattelandse praktyk veelal 'n eenmanspraktyk is, en dat die geneesheer se pligte hom nie die tyd laat om oor lang afstande vergaderings van die Vereniging by te woon nie, laat staan nog om hom beskikbaar te stel vir verkiesing op liggame waar ons saak gestel kan word.

Die feit dat die reisgelde van 35 sent per myl vir die besoek van pasiënte van die dae van die perdekar af nog nie verhoog is nie, dui vir my verder op die koue belangstelling van die Federale Raad in die wel en wee van die plattelandse lid. Die nog veel laer reisgeld in die geval van goedgekeurde mediese skemas maak totaal nie voorsiening vir die verlies van konsultasietyd nie.

Ek hoop dat hierdie brief deur ander met soortgelyke strekking opgevolg sal word.

B. P. J. F. Coetzee

Kerkstraat
Dealesville
12 Julie 1961

1. Van die Redaksie (1961): S. Afr. T. Geneesk., **35**, 557.